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SEPTICAEMIA*

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Introduction: "When we are born, we cry that we have come to this great stage of fools." Mankind develops, matures and declines more or less intimately associated with various proved septic microorganisms, since the latter for practical purposes are ubiquitous. Self preservation from septicaemia primarily depends upon the resistance of the host, the dose and virulence of organisms accidentally or otherwise inoculated into the circulation.

HISTORY

Hippocrates recognized complicated wound infection.

John Hunter, 1774, discussed the factor of suppurative phlebitis.

Virchow, 1856, credited metabolic abscess to infected emboli.

Pasteur taught puerperal sepsis was of bacterial origin, confirming the ideas of Oliver Wendell Holmes and Semmelweis.

Leube, 1878, described cryptogenic septicaemia.

BACTERIAL ETIOLOGY

In order of their frequency: Streptococcus, staphylococcus, pneumococcus, colon bacillus, gonococus, meningococcus, anthrax, gas bacillus, pyocyaneus, diphtheria and influenza.

PORTAL OF ENTRY

Skin lesions rank first, puerperal origin second and focal infections third; e.g. the latter includes tonsils, sinuses, mastoid, respiratory and genito urinary tract, the intestines, liver, gallbladder, bone and teeth.

PREDISPOSING CAUSES

Arise by virtue of lowered resistance, as exposure to cold, malnutrition, fatigue and chronic disease. However, the dose and virulence remain important factors, besides certain bacteria may become resistant to the individual's protective agencies, and become aggressive, while many less toxic members of their species would have succumbed. Flexner states a bacteriaemia is demonstrable in 20 per cent of the cases of chronic kidney and heart disease. Preagonal diminution of immune bodies permits 84 per cent of all deaths to result with some type of terminal infection. It is therefore evident the preexisting condition of the host is a primary factor in allowing septicaemia to develop.

DEFINITION WITH QUALIFICATIONS

Septicaemia is best defined as a symptom complex, since the symptoms and complications result from the bacteriaemia; besides, it is not strictly speaking a definite disease entity, inasmuch as the etiology is variable. Transient bacteriaemia is very common, but in practice rarely produces constitutional phenomena, save in those disease entities in which the bacteriaemia appears early but eventually ceases in the natural history of these diseases by virtue of antibody formation, e.g. typhoid fever, pneumonia, anthrax, malta fever and plague. Fortunately, such disease entities

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develop other definite characteristic clinical and laboratory phenomena different from pyogenic septicaemia that will help make a differential diagnosis. These and similar disease entities will not enter into my discussion, nor these other purely local infectious processes of haemotogenous origin, e.g. perinephric abscess and gonorrhoeal arthritis, since the normal circulating blood is capable of freeing itself of these bacteria. Generally speaking, the modern concensus of opinion holds bacteria are unable to multiply in the blood stream save in many terminal states, after most anti-bacterial mechanisms have broken down; except possibly in the stagnant capillaries of the liver, spleen and bone marrow. (Tileston.)

Bacteriaemia in a limited degree frequently occurs without symptoms or material damage and ends in complete recovery; but when resulting from some focal infections, that by virtue of a sensitization permits these pathogenic bacteria to reproduce in distant places and develop destructive lesions, e.g. pneumococcic meningitis and arthritis, we recognize a well known exception and that often proves itself obstinate.

Pathogenesis

We believe a small dose must be oft repeated, or a massive toxic dose must have entered the blood stream, e.g. following ill-advised surgery in streptococcic lymphangitis. The natural normal defenses appear competent to dispose of a non-repeated small dose, while polymorphoneuclear leukocytes and the reticuloendothelial system notably those cells in the liver and spleen, are prone to come to the rescue with efficient phagocytosis. In practice we have observed septicaemia commonly arises from a lymphangitis under conditions where the lymphatic glands are inadequate to block fully the ingress of repeated doses of toxic bacteria. Likewise it often arises from a septic thrombophlebitis persistently discharging septic emboli, that in turn cause metastatic pulmonary abscesses, which eventually spill their contents into the blood stream, either too frequently or in too large quantities for the natural defense processes to effectively destroy or eliminate these doses.

Morbid Physiology

Early symptoms arise from the toxaemia, haemolysis and embolic processes with deranged systemic functions. We are taught if this picture is not early recognized and successfully treated, multiple visceral degenerative processes will eventually ensue and the case becomes too advanced for any known successful management.

Morbid Anatomy

The spleen enlarges, cloudy swelling with fatty changes in the viscera eventuate with numerous foci of necrosis. The serous membranes and joints are especially vulnerable. Nephritis, metastatic abscesses and infarcts are common. Endocarditis occurs in 50 per cent of staphylococcus cases, 25 per cent from pneumococcus and only 8 per cent from streptococcus. Various skin eruptions are common and may be petechial, papular, pustular, bulbous or hemorrhagic. Progessive anaemia follows unless transfused. Haemolytic jaundice may eventuate. Leukocytosis, with excess of polymorphoneuclear cells, ranges from 10,000 to 50,000 per cubic millimeter and when persistently below 10,000, is considered of serious prognostic omen. Empirically multiple accessible external metastases augers a favorable immunological phenomenon.

Mortality

Mortality is somewhat higher when over 25 years of age; streptococcus 66 per cent, staphylococcus 71 per cent. The number of colonies from cultures are not especially prognostic, but the outcome depends largely on whether or not the etiological focus is amenable to competent and intelligent surgery; secondarily, on the availability of specific sera, and to no small degree on competent general management.

Clinical Types of Pyogenic Septicaemia

1. Fulminating: Results from an overwhelming dose of virulent bacteria in patients with low resistance. Usually fatal in a few days regardless of surgery and medical treatment.

2. Acute: Normally runs a course of a few weeks and may recover if the focus was amenable to surgery and the necessary specific serum was available.

3. Subacute cases: May be indefinite in their course. Death, when it results, is

from complications, because bacteria appear to acquire a super-resistance or results from ultimate degenerative pathology of important viscera. The acute and subacute types may assume a typhoid state, resemble meningitis with marked delirium or coma, or may closely simulate acute rheumatic fever with endocarditis and polyarthritis.

Definition From Concensus of Opinion

Pyogenic septicaemia is a morbid condition resulting from the presence of pathogenic bacteria and their associated poisons in the blood stream. The bacteriaemia persists because bacteria are continuously being fed into the circulating blood. In all cases, septicaemia is a disease of surprising vicissitudes and uncertainties.

Diagnosis

The probable etiological history offers us our first dependable clue. Positive cultures are possible, along with the early toxaemic symptoms. The blood specimen is best taken during the rising stage of the fever or chill. Full cooperation with a well equipped medical technician is indispensable for accurate information and proper typing of positive cultures. Accuracy of leukocyte counts and percentages of each type is likewise mandatory. The onset varies but may be acute, with chills, irregular fever, stupor, delirium or coma. The pulse is rapid and systolic cardiac murmurs are common. Some have anorexia, nausea, vomiting, febrile albuminuria and retinal hemorrhages. High leukocyte count with preponderance of polymorphoneuclears are expected. Later, embolic processes with metastatic abscesses, arthritis, osteoarthritis, etc., clears the picture sufficiently for tyros. Irregular fever and chills, associated with drenching sweats and a rapidly developing anaemia with emaciation, are almost pathognomonic.

Differential Diagnosis

1. Typhoid fever presents a relatively slow pulse, leukopaenia, dull facies, rose spots in crops and intestinal hemorrhage.

2. Rheumatic fever: shifting, non suppurating arthritis, pain yields to salicylates; no emboli or enlarged spleen.

3. Malaria: plasmodia demonstrable and yields to quinine.

4. Miliary tuberculosis: probably demonstrable focus, dyspnoea, cyanosis, tubercles in the choroid and a lymphocytic leukopaenia.

5. Likewise pyelitis, Charcot's fever, perinephric abscess, undulant fever and Hodgkin's disease have sufficient clinical characteristics, along with laboratory findings, to render their diagnosis reasonably certain; however, we fully realize septicaemia may complicate or co-exist with other disease entities, and not uncommonly arises from ill advised surgery producing favorable entrance and spread of pyogenic bacteria to the blood stream. The clinician has learned when he observes small vesicles presenting a red halo and rapidly becoming pustules, the staphylococcus is the probable etiologic factor. The pyocyaneus cases present hemorrhagic spots becoming necrotic and suppurating at the center. Streptococcic invasion rarely obtunds the mind early, but victims are mentally alert and scrutinize the physician's attitude unusually closely.

Treatment

Physicians and surgeons should invariably endeavor to aid the patient intelligently, and not at any stage to hamper recovery through natural processes by ill advised exhibition of questionable and unproved remedies or procedures. They both fully realize progress in the management of pyogenic septicaemias has already, far too often, been retarded by tradition, superstition, half truths and untruths. They have also learned their lesson and fully realize symptoms ordinarily antedate serious structural changes and are therefore alert to establish the diagnosis and use intelligent management before irreparable damage occurs. The intelligent physician prefers to cooperate with a trustworthy surgeon and competent medical laboratory technician, before he outlines specific procedure or finally evaluates his prognosis. He furthermore fully realizes that immunity, natural or acquired, is never absolute but rather tends to vary with the individual at different times. After having consulted all his available authorities he also regretfully concludes our factual knowledge is still problematic, and the opinions of authorities still vary concerning the modus operandi of immunological processes.

However, the practical, conservative physician does know empirically that the successful management of pyogenic septicaemias depends primarily on his being able to modify such morbid physiology that exists in accord with what is normal in order that he might produce unfavorable environment for these bacteria; rather than implicitly follow his desire to kill or neutralize this type of bacteria with over advertised commercial specific drugs or serums.

General Principles of Managewent

1. Intelligent and competent surgery; when possible, remove, drain or isolate the distributing focus.

2. Ample rest and comfort, sedation, oxygen and vitamins when indicated.

3. Observe closely the intake and output of fluids, with minimum quantity per day of 3,000 c.c. as necessary, fluids parenterally as indicated.

4. High caloric diet relatively rich in proteins, symptomatic and expectant treatment to aid digestion and reasonable elimination.

5. Multiple small transfusions, with whole blood or serum, depending on existence and type of the anaemia; use immune donors when obtainable.

6. Administer suitable stimulants when indicated, best selected according to the presence of morbid physiology of the various systems, e.g. cardiovascular, respiratory and nervous system.

7. No antipyretics should be used, but hydrotherapy for fever moderation as well as cleanliness and comfort. We regard fever as a defense mechanism; realizing it will increase metabolism, change the albumin globulin ratio favorably to fibrinogen and globulin, lessens the suspension stability of the blood and decreases platelets, but increases leukocytosis, water retention and favors agglutination.

Special Measures for the Specific Infection

1. Staphylococcic Septicaemia: We believe meticulous carrying out of the general principles already enumerated with judicious use of transfusions, offers about all that has proved advantageous. However, alert, competent and radical surgery should be emphasized. Thrombophlebitis about the etiologic focus is preferably removed in toto, also ligation of veins likely to carry infection when feasible. Serums and bacteriophage are neither condemned or condoned by the authorities, but if used, the usual sensitization tests should be made before their use and we have learned by experience unfavorable reactions are less apt to occur if they are administered while the patient is under anesthesia.

2. Streptococcus Haemolyticus: Since the various strains of streptococci are numerous and not fully worked out, the use of specific sera is problematic, but favorable agglutination tests of cultures from the patient will help determine if those serums available should be administered.

The reasoning is analagous to use of serums for pneumococcic infection, except we have less definite information concerning the types involved. Justification in the use of polyvalent serum exists, particularly if improvement ensues with the first few doses, it may be advantageously continued, but if not, discontinue its use and change to some other company's product. It is very easy for one's wishes to overrule his judgment in the use of serums, since we are prone to forget nonspecific serums for the case in hand might readily prove harmful. Pyogenic bacteria require an antibacterial serum and materially differs from the use of antitoxic sera that have proved so effective, e.g. in diphtheria and tetanus which act by neutralizing toxins, comparable to the well understood acid base neutralization in chemistry. Sera, to overcome bacteria with endotoxins must also contain ingredients comparable to those existing in immune serum, i.e. praecipitans, agglutinins, bacteriocidal and bacteriolytic substances which will in some way subject this particular strain of bacteria more readily to phagocytosis with ultimate destruction or neutralization by proteolytic enzymes.

The anaerobic streptococcus putrificans so common in septic abortions, when proved present, may be rendered less harmful by gently dilating the cervix and thoroughly washing out the uterine cavity with oxydizing agents as taught by Dr. Otto Schwarz, nevertheless is disapproved by other equally prominent obstetricians.

Surgery

When dealing with known streptococcic pathology, e.g. lymphangitis, the more conservative the surgeon the better, the existence of fewer avenues for dissemination with nature's protective exudates intact outweighs the advantage of free drainage from early incision even though done with the cautery.

Sulphanilamide

At present has popular approval and does seem promising, but as yet we are not fully informed concerning its contra-indications. The dose recommended as 1 Gm per nine Kilos daily, best given in divided doses per oram, or parenterally. Direct application, or injection into cavities as empyema is permissible. Ninety per cent show cyanosis to some degree; nausea, dizziness, tinnitus and diarrhea may develop. Rarely nitroid reactions occur, e.g. dermatitis, eczema, hepatitis, or optic atrophy. "It produces in some patients a stimulation and in others depression of leukopoetic activity." (Krache.)

This drug should not be administered so promiscuously as is done at present, and it is safer to hospitalize such cases so the proper dosage may be maintained, i.e., approximately 10 M. Gm to 100 c.c. of blood, proved by laboratory methods. Besides, take frequent white counts to avoid serious leukopaenia and agranulocytosis, also make tests for N.P.N. and when found below 15 or 20 milligrams per cent, discontinue the drug. It is thought by some that the newer derivatives of sulphanilamide, e.g. neoprontosil produce less toxicity and are possibly more efficacious in bacteriocidal action.

Many other antiseptic drugs and dyes have been in the past highly recommended, but to administer them in the hope of performing miracles, is not at present tenable; since without exception they have been weighed in the balance of experience and found wanting.

Autogenous vaccines and bacteriophages are permissible particularly in subacute and chronic infection and both have the indorsement of some reliable authorities.

Artificial fever by foreign proteins or other methods is not to be considered because the extreme continued hyperpyrexia necessary to kill the pyogenic bacteria would be lethal to the host.

Theoretical and Experimental Consideration

If the physician institutes proper management, it is necessary for him to understand the patient and be able to visualize all co-existing and correlated pathological conditions, with their morbid physiology, as well as to diagnose and evaluate the status praesens from the superadded disease entity. He furthermore fully realizes and takes cognizance of the fact that constitutional and hereditary factors of any human being cannot escape the germ plasm of which they are made and furthermore, environment might mitigate but it can not change the general trend; also his ability to be of service will depend more on bringing about favorable changes in the patient's reactions, rather than theoretical modification of the bacteria. In some respects all patients appear alike in their morbid reactions, while in others, e.g. immunological reactions, they may be vastly different, as observed when allergic states present themselves, apparently out of a clear sky, with explosive exudates and outpouring of fluids. These allergic problems here presented have as yet not been clearly explained or very satisfactorily met by the symptomatic relief afforded from adrenalin, ephedrin, atropine, calcium, auto transfusion, etc. This phase is mentioned merely to emphasize the hazards that may arise with serum therapy and places responsibility on their giver to the extent that he should be familiar with sensitization tests, use due caution concerning proper desensitization in susceptible individuals; be prepared to meet anaphylactic reactions, thermal reactions and serum sickness intelligently.

Such research tests that are requested should, in my opinion, be evaluated somewhat on your confidence in the technician; non-medical technicians are at times too positive, probably because they fail to realize fully dead specimens may react differently from what they would were they yet bioplasm, besides they may not fully comprehend all the phases of the problem presented to the physician. A comprehensive consideration of problems concerning pyogenic septicaemia brings us face to face with the parting of the ways

from strictly scientific procedures and proved facts, to the reasoning by analogy followed by experimental dreamers. The facts are, with reference to pyogenic septicaemias, that if truth is ever to disrobe herself entirely, her underclothes will most likely be in the proud possession of an experimental dreamer.

Dr. Fred T. Cadman of Winnipeg, Canada, has to his credit the following:

He kept a large colony of rabbits injected twice weekly over a period of months with pyogenic bacteria. Whenever he had a case of pyogenic septicaemia, he made a vaccine with a heavy culture from this patient; this process took only 48 hours, including injection of this specific vaccine into a few of his prepared rabbits and after 24 hours, bleeding them from the heart and using this serum for the patient, claiming the titer is very high in antigen. For the ambocepter he makes use of commercial serums, besides furnishes more compliment from human serum, claiming it acts better than transfusion with whole blood by seeming to activate further the patients compliment, that has been found by him to drop materially after the first transfusion of whole blood, and is not much increased by subsequent transfusions. So far, Dr. Cadman has treated 100 cases in this manner and reports a mortality of only 15 per cent.

O. H. Robertson, M.D., observed that in experimental pneumonia the animal always dies unless there exists macrophage immune bodies in their lungs and their blood is also capable of destroying the pneumococci. While we do not fully understand the reticulo-endothelial system, we are taught its cells develop from special types of endothelium located in various organs, e.g. spleen, liver and bone marrow, also from the wandering cells of the reticulum. We think the monocytes are these cells that have entered the circulation and are so efficient in disposing of broken down red cells, which products finally eventuate into bilirubin, iron and globin. Besides exercising an all important phagocytic function, the reticulo-endothelial system in some way influences metabolism as exemplified by Gauchers and Nieman-Picks disease as well as the Hand-Christian Syndrome and Xanthomatosis.

Most authorities grant active phagocytosis is a major part of our immunological reactions and the presence of opsonins is essential in preparing bacteria for the phagocytes. These phagocytes seem eager to ingest the crudest of food substances, notably particulate matter and the colloids. Moreover, they are able to digest or render most toxic substances harmless through the action of their proteolytic enzymes; at all events, no metabolic or functional disturbance follows their death.

Metchninkoff went so far as to teach that both immune bodies and compliment are derived from the leukocytes. Whenever the reticulo endothelial system becomes partially blocked with colloids, nature proceeds to unblock it by a further rapid hyperplasia, which produces more platelets and fibrinogen thereby shortening the coagulation time.

Diaz claims this system can be stimulated by the use of 10 c.c. of 1 per cent Congo-red in distilled water. The question naturally arises, are we sleeping on our rights in neglecting to bring this important reticulo endothelial system actively to our assistance? Or would we by so doing, wave a two-edged sword capable of producing unknown conditioned toxicities, analogous to harmful acquired allergies, or idiosyncrasies? At all events, reasoning by analogy, we do realize there are conditioned reflexes of the nervous system.

Human nature is most sorely tried when we physicians are forced to surrender to the inevitable; after due consultation and with conscientious prolonged effort to rescue our friends from pyogenic septicaemia. There arises in me a temptation somewhat analagous, I imagine, to the state of mind of a broke merchant endeavoring to recoup his losses by a last frantic effort in staking his possessions on the possibility of pyramiding in a freakish market. Like his chances, the possibilities of cure are scant, but when in this frame of mind, I still at least talk about fixation abscesses, replacement transfusions and unblocking the reticulo-endothelial system.

Conclusions

We are at present possibly overtreating our septic patients; since the existing potential sources of septicaemia are so nu-

merous, it is reasonably safe to conclude natures methods cure all this multitude of those cases that are undiagnosed. Intelligent and competent surgery is imperative, specific serums are advantageous, and general principles are always helpful, but when doubt arises in your own mind, hesitate and don't unwisely interfere with natural processes.

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Discussion

Dr. Horace Reed: From the moment we are born until death overtakes us, we live constantly in close proximity to danger. When we contemplate how narrow the threshold really is, we may easily become morbid. With coolness and precision, so far as our knowledge permits us to go at this moment, the essayist has discussed the danger to which I refer, namely, septicemias. There are only two factors which serve to protect us from that danger. One is anatomic, or more strictly hystologic, because the barriers which every moment of our lives protects us from the invasion of the pathogenic organism is microscopic in dimension. The other factor may be called physiological, or perhaps more strictly speaking, biological. A substance which also may be infinitesimal in quantity, namely, antibodies.

Dr. Lamotte has presented us with a thesis on the subject of septicemia. So far as I have been able to determine, he has presented us nothing but facts which are historical, or with theories which have been thoroughly proven, and when he has entered the field of speculation he has been judicious and conservative. In other words, it is difficult for one to add to or disagree with what he has presented, so thoroughly has he considered every phase of the subject. In doing this he has exhibited a profound insight into the art of the practice of medicine.

The bacteriologist more than a half century ago opened the door which let in the light by which we could see the causative agents of septicemia, namely, the "ubiquitous microorganisms," and since we have known the cause, we have been giving more and more attention to the laboratory in searching for the cure and, correspondingly, more and more have been neglecting the patient. Long before there was a science of bacteriology, physicians treated septicemia. There were no laboratories, hence, the physician himself treated the patient. Since laboratories have become so common, the tendency is to neglect more and more the patient and treat the disease.

Dr. Lamotte has demonstrated in his presentation how important it is not to neglect the patient, who, because of his affliction, is possessed with a morbid power of reasoning. The products of that which affects him make him mentally sick. Well may we pause to inquire as did Macbeth, "physician, canst thou not minister to a mind diseased?" Truly this art in the practice of medicine is being sadly neglected. There is no panacea or true specific in the treatment of septicemia, and the essayist has made this clear. Of course, in covering the subject of treatment, which has been done so thoroughly, he had to resort to brevity.

In discussing treatment, therefore, one can only hope to add to the value of this paper by emphasizing those points, the value of which are so well proven. The first of these that I would emphasize would be *rest*. The value of rest in treatment of septicemia, rest which is physiological, would be a subject well worthy of a thesis of itself. Nearly a century ago, Hilton delivered a series of lectures on rest and pain which, when collected, form a good sized volume. Every physician who assumes to treat anyone afflicted with an infection, it matters not what the causative agent may be, should have read Hilton on "Rest and Pain." In overwhelming infections produced by those organisms which are commonly found in what we call septi-

cemia, the value of rest should be emphasized more than anything else, for without it all other methods of treatment will surely be disappointing. With rest as the foundation upon which to build the management of a patient with septicemia, the outcome will be less likely to be disappointing. But even with rest thoroughly carried out, and all the other known and tried adjuncts in treatment, septicemia remains a dangerous disease.

Next to rest is nourishment. If the patient is to combat successfully the invasion of the hordes of organisms and their nefarious cohorts, the virulent toxins which they generate, he must be well sustained. Food of appropriate kind furnishes fuel by which he may develop reserves to carry on the fight. Well may it be said that the patient fights on his belly as does a successful army.

Next in the line of treatment may be mentioned repeated, even though small, blood transfusions. Sufficient time has elapsed since transfusions have been used, and the results obtained have proved beyond a doubt that this in many cases is most beneficial. Theoretically, at least, if a donor could be used who has successfully overcome and fully recovered from this disease, the results should be more beneficial.

Dr. Lamotte has not overlooked the new panaceas, but we are pleased to note with what caution and conservatism he mentions them. Some of us "whose blood and judgment" are not so well commingled, would have blatantly described some of the newer remedies, particularly sulfanilamide and its allied chemicals. One of the discouraging things in later years is to note how increasingly gullible we as physicians are becoming. How many cure-alls of biologicals and serums have literally blazed across the sky in the last 20 years, only to disappear behind the horizon of gloomy disappointments. Who of us can still remember one of the earlier ones which was heralded as a most outstanding cure-all in infectious conditions, namely phylacogen. I, personally, missed by a hairsbreadth becoming one of its most ardent supporters. Having been asked to see a patient in consultation who was suffering from childbed fever, in whom the doctor had employed every known facility or remedy which up to that time had produced no apparent improvement, except phylacogen, I recommended it, even though I had never personally employed it. However, I had read the very exciting and alluring literature which was in all the medical publications at that time. Fortunately, or unfortunately, such was the demand for the product that it was not available. Three or four days later the patient's temperature dropped to normal from which point her recovery was rapid and uneventful. Had my pet remedy been available, what a wonderful cure it would have produced, assuming, of course, it did not of itself produce harm.

Sulfanilamide and its kindred products are just at this time blazing through the skies as the great cure-all of most of the known ills caused by infection. Let us hope that in the end the effect of the remedy itself will not prove to be as bad, if not worse, than the diseases for which it is being so injudiciously used. There is no doubt that it gives hope of the coming of a very valuable addition to our armentarium, but because it does give promise, let us jealously guard it against abuse until its possibilities have been made known.

Common Cold May Cause Infections In Sinuses

The common cold can cause infection in the sinuses, other parts of the respiratory tract and ears, Sidney N. Parkinson, M.D., Oakland, Calif., says in The Journal of the American Medical Association for January 21.

Nasal congestion during a cold interferes with circulation about the openings of the sinuses. This increases swelling and congestion within the sinuses and permits accumulation of mucopus which the hair-like projections in the respiratory tract are unable to remove. This complication is unfavorable to tissue defense.

"The purpose of local treatment during acute infection is ventilation in order to improve drainage," the author says. Shrinkage of the nasal mucous membranes with drugs opens the air passages. Free drainage then takes place if in the process of ventilation the hairlike drainage mechanism has not been damaged. This is why the selection of a physiologic drug is so important. Ephedrine in Locke's solution or its equivalent constitutes an efficient harmless agent for shrinkage.

The drug best reaches the membranes of the air passages with the patient lying on his side with his head bent downward exactly sidewise, using the shoulder as a fulcrum.

After from three to five minutes the head is rotated to face down to permit the nasal contents to escape from the nostrils. The head-low posture permits all important structures within the nose to come in contact with the medication and obviates any injury.

Remarks on Treatment of Dacryocystitis*

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It is not my intention to present anything new in the treatment of inflammation of the tear sac, but the subject has not been reviewed before this section for some time and perhaps a few remarks concerning it are thereby appropriate to promote discussion and this explanation might serve as an apology for the rather didactic remarks to follow.

Dacryocystitis is somewhat unique in that the usual procedure of an acute process incompletely healed resulting in a chronic state of inflammation is exactly reversed. Acute dacryocystitis results from a chronically infected sac, and we should therefore discuss the chronic sac first.

"Chronic dacryocystitis is an inflammation of the mucous membrane of the lacrimal sac due to impeded outflow and consequent stagnation of the tears." This is the sum and substance of the pathology of this condition and is taken verbatim from Fuchs. The nasolacrimal duct becomes narrowed or completely occluded by cicatricial contraction, congenital conditions, obstruction by mucoid or mycotic plugs of material, or by extrinsic pressure by tumors in the region of the sac; very frequently polyps in the nose obstruct the terminal opening. This leads to stagnation and infection, the same as in a kidney pelvis with ureteral obstruction or a gallbladder with cystic or common duct obstruction. It is a truly chronic condition and lasts for years. Actual suppuration may be present in the early stages but soon gives way to a mucoid type of secretion or in the late stages merely watery tears.

The obstructions seen in young children due to congenital conditions are usually due to groups of epithelial cells within the duct or thin membranes, most often in the region of the nasal opening beneath the inferior turbinate. Pressure over the sac by the physician together with instruction of the mother in the procedure to be repeated several times daily empties the sac through the puncta and so prevents stagnation; but it also is frequently successful in breaking down the obstruction by increasing the pressure through the nasolacrimal duct and so making the duct patent. I seldom have found it necessary to pass a probe in an infant when this procedure is thoroughly carried out. General anesthesia is, of course, necessary for this procedure.

Chronically infected tear sacs are always dangerous. The bacterial content can be any of the pathogenic organisms found in the conjunctival sac; they are opportunists, awaiting a break in the surface epithelium to begin their work. I was struck by the large number of references in the foreign literature to mycotic infection of the tear sac and the scarcity of it in English articles. We perhaps do not study the bacteriology of the sac as well as we should. Tuberculosis, trachoma and syphilis may be harbored there. The pneumococcus is a frequent offender; hemolytic staphylococcus and streptococcus are frequently present. Routine conjunctival smears should include firm pressure over the lacrimal sac to express some of its contents, for many times an apparently negative conjunctiva contains pathogenic organisms in its sac.

I have spoken before of the procedure advocated by Green with reference to the lacrimal sac in cases of corneal foreign bodies. In those cases in which the foreign body has penetrated through Bowman's membrane and involves the true corneal substance we know that healing will be delayed for 36 to 48 hours. Here is an excellent opportunity for organisms present in an infected sac to set up infection and result in a severe corneal ulcer. In these cases one makes firm pressure over the tear sac, and if it is at all suspicious of increased content, one fills the canaliculus

*Read before the Section on Eye, Ear, Nose and Throat, Annual Meeting, Oklahoma State Medical Association, Oklahoma City, May 2, 1939.

and sac with White's ointment, using an ordinary Record syringe and lacrimal needle for this purpose. The antiseptic is dissipated slowly and two fillings usually will carry one beyond all danger of infection. It is my belief that this procedure is a good safeguard against the possibility of a severe corneal ulcer following corneal injury.

It is of course unnecessary to discuss the absolute need of clearing up any chronic infection before all intra-ocular operations.

Non-surgical methods of treatment include probing and irrigating. A sound should always be passed gently and cautiously because of the danger of creating a false passage. Too much should not be attempted at one sitting. I am sure that we younger men are not as adept in this procedure as the older eye doctors are for it apparently is not used as frequently at this time. Nevertheless an attempt should be made to re-establish patency by judicious use of the probe, and if successful, passage of the sound at intervals will often solve the problem.

Every known antiseptic solution has at some time, in some hands, been used as irrigation in this condition. In the duct with partial obstruction it is my opinion that normal saline is as good as any, though I have used many of the mercurial preparations and feel that Pregl's iodine solution has been of definite added benefit in some cases. Many patients will refuse surgery and irrigation must be continued.

In the case of complete or almost complete obstruction, not overcome by probing, surgery is indicated to relieve the constant epiphora and danger of infection of other ocular structures, particularly the cornea.

Removal of the sac has been practised for many years and every man was trained in the landmarks of anatomy and steps of the procedure. But this apparently simple extirpation more often than not turns out to be a formidable one. Bleeding can be severe, especially when one is working in the region of the superior portion of the sac and it is easy to leave a small portion of the sac lining in this area with a poor end-result. After its completion only one of the benefits has been accomplished however, that is, removal of the danger of infection. Annoying epiphora is still present and in most cases many years are still ahead of the patient and the constant tearing often results in excoriation of the skin, a very annoying condition.

In choosing an operation upon the tear sac, one desires to restore the physiological function, i.e. to allow the free drainage of tears from the conjunctival sac, through the canaliculus and tear sac into the nose.

The method of treating chronic dacryocystitis by creating an artificial passageway into the nose is an ancient one, for Celsus and Galen before the third century treated this condition by plunging a red hot cautery through the lacrimal bone into the nose. Refinement has taken place through the work of Montaigne, Dupuy-Dutemps, Polyak, Toti, West, Arruga, Mosher, Poulard and many others. All have as their object to drain the contents of the lacrimal sac directly into the upper nasal cavity. In order for the operation to be successful, there must be some of the lacrimal sac remaining and there must be an opening from at least one of the lacrimal puncta into the tear sac. This operation can not be performed therefore after removal of the sac, whereas if unsuccessful, removal of the sac is possible as a last resort.

An intranasal approach as in the West operation can be used, but I have had no experience with this technique. The external operation, particularly that of Toti, modified by Mosher, is in my opinion more easily performed and has been successful in my cases. Marked nasal deformities such as deviated septum should be corrected before the operation and fracturing of the middle turbinate to give more room in the meatus above it often helps. It can be done under local anesthesia or in combination with avertin in cases where this is indicated. Failures by either technique are usually due to the formation of dense scar and granulation tissue in the nasal opening. Another complication that must be borne in mind is delayed and severe hemorrhage which may occur from the anterior ethmoidal arteries.

Acute dacryocystitis is a cellulitis of the connective tissue surrounding the sac, a result of infection from a chronic one entering by way of a break in the mucous

membrane. It may therefore be a complication of probing that results in a false passage. Oftentimes it occurs with no preceding manipulation and in these is probably due to ulceration or necrosis of the lining. The phlegmon goes on to abscess formation necessitating incision. Measures to abort this abscess formation should be taken early, for incision almost invariably results in fistula formation which is difficult to close. Sulfanilamide in cases of appropriate bacteriology should help in some cases and if used early, X-ray irradiation can be used.

After the acute inflammation of the tissues have subsided, the underlying chronic sac must be dealt with for stenosis of the lacrimal duct was originally present. In many cases the fistula remaining will not close until the flow of tears into the nose is re-established. One of the procedures mentioned must be carried out and very frequently surgery, either extirpation or dacryocystorhinostomy must be done.

In conclusion, dacryocystitis is a condition presenting many difficulties in its management. The improvements in the technique of dacryocystorhinostomy has made its management much easier for the ophthalmologist and patient alike.

Diverticulum of the Bladder*

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Diverticulum of the bladder is a condition which has not received clinical recognition until recent years. It was known to MORGAGNI, and several cases were reported as internal cystocele by CHOPART and CIVIALE. Already in the early 19th century the associated causal relationship of prostatic hypertrophy and increased intracystic tension was recognized.

The modern era began with the introduction of the cystoscope, and in 1901 DURRIEUX reported 200 cases collected mostly from postmortem autopsies. Later, radiology was applied to urology with complete visualization of the diverticula by means of cystograms. PEAN and CZERNY were the first to operate for this condition. YOUNG, in 1906, reported successful operation cases, and described new technique, which, with slight modifications, has stood to this day.

The diverticulum itself is a sac communicating with the vesical cavity and comparable with the condition seen elsewhere in the colon and pharynx. Diverticula may be large or small, single or multiple. Cases are reported of the sac containing more than a gallon of urine. As a rule, the orifice is quite small and out of proportion to the size of the pouch.

In the female the diverticula are not common. They may be associated with old standing pelvic inflammation with adhesions. These are traction diverticula, and may develop in any area of the bladder.

The pathological changes noted in diverticula of the bladder are atrophy and progressive chronic inflammation which may lead to cancer. The wall is tough and sometimes almost parchment-like. The inner surface is lined with a fine velvety membrane. Phosphatic calculi are very frequently found in the lumen of the diverticula. In the microscopic examination the mucous membrane is atrophied and may show extensive areas of superficial erosion. The bulk of the wall consists of fibro-granulation or fibrous tissue. Strands of muscle tissue are present. In the early stages the muscle may be well marked, but in old standing cases the muscle is broken up and may be found in many parts of the wall. A fine scar tissue is frequently noted, particularly under the mucous surface. There is very considerable infiltration of the wall with round cells.

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This is most prominent under the mucosa, and, in association with the large vessels in the outer layers, eosinophil cells are very noticeable. The fine capillaries near the surface are engorged and small extravasations of blood are frequent.

The origin of these diverticula is uncertain. By many it is held that, although they may not manifest themselves till late in life, they are aways due to congenital predisposition. It is pointed out that they most frequently occur at certain places in the bladder which are developmentally weak, namely, just above the ureteric orifices, in this position being often symmetrical, or less commonly just behind the interureteric bar. In the course of routine cystoscopies it is not rare to find small recesses in the mucosa above and behind the openings. These may well be the precursors of true diverticula which develop later in life when some obstruction has occurred to the outflow of urine from the bladder. Another view is that they are formed from accessory ureteric buds.

When diverticula occur apart from any obstruction to the outflow of urine from the bladder they must certainly be congenital; but even when obstruction is present it may well be argued that this has merely made obvious the presence of a pre-existing pouch by causing a great increase in its size or perhaps an infection of its contents. It has been, however, proved that true diverticula can also result from obstruction alone, such as a narrow urethral stricture lasting for several years.

Diverticula have been produced experimentally in dogs by a combination of increased intravesical tension and localized injury to the bladder wall. The production was facilitated by the onset of infection. Infection easily develops since the urine stagnates in the diverticula.

The more moderate-sized diverticula do not, as a rule, give rise to symptoms unless they become infected. The larger diverticula, even before infection occurs, usually give the patient sufficient discomfort to cause him to seek medical advice. A quite common sign of bladder diverticulum is hematuria, which may be due either to the inflammation to the sac, or to calculus in it, or the rare association of tumor with diverticulum. Hematuria, of course, may be due also to the coincidence of an enlarged prostate.

More frequent than hematuria are the troubles of micturition. After voiding there is usually a second stream of foul urine which, however, occurs also in tabetic affections of the bladder and urethra, and is not pathognomic of vesical diverticulum. Frequency of micturition is often met with, and this common symptom is usually explained by the cystitis which so often results from the presence of the pouch; in other cases it may be due to an enlarged prostate. Difficulty of micturition often results from the association of an enlarged prostate, or stricture, but may be produced by the diverticulum itself. Retention of urine may result if the diverticulum interferes with the proper action of the trigonal musculature.

Pain is a symptom usually present when inflammation has developed. It is usually related to urination. Sometimes the diverticulum itself may cause pain which is felt particularly in the groin, and to a less degree in the perineum. Such pain may probably be due to the forcing of urine into the diverticulum and the stretching of the adhesions which, as is so often the case, surround the diverticulum.

The chief complications of bladder diverticulum are: 1. Involvement of a ureter. 2. Calculus formation. 3. Abscess formation within the diverticulum. 4. Neoplasm within the diverticulum. 5. Diverticulum in a hernial sac. 6. Compression of a ureter with resulting hydronephrosis and renal infection. 7. Constipation from pressure upon the rectum.

The most important aids in diagnosis are: 1. Cystoscopy. 2. Cystogram. Cystoscopy readily identifies the orifice of diverticula of any size. The location where it may be missed is at the summit of the bladder, at the urachal attachment. Some rough estimate of the capacity of a diverticulum may be had by passing a ureteric catheter into the sac. The most accurate information may be obtained by use of the cystogram. The bladder is filled by catheter and syringe or funnel with a sterile solution of 10 to 12½ per cent sodium iodide. It is very useful to watch the filling under a screen, but is not necessary. One hundred and fifty to 200 c.c. of fluid is usually sufficient and then take cysto-

grams in whatever positions are best calculated to show the form and extent of the pouch. Other radiograms should be taken after the catheter has been allowed to drain, or the patient allowed to micturate. Radiograms should also be taken apart from cystography, for, as mentioned, they may reveal calculi whose existence would otherwise not be suspected.

According to the cystogram, diverticula may be separated into two groups:

1. Non-retentive.
2. Retentive.

In the first group, which empty fairly well with the act of micturition, and in which there is little or no retention within the sac, no treatment is required. After relief of the raised intracystic tension the pouch tends to egress. If the bladder is for any other reason opened, it will be good to stretch the orifice of the sac with the fingers to enhance drainage into the bladder.

In the second group there is often considerable retention, usually several ounces. This is typically the large, flaccid, fibrous sac with a narrow, rigid orifice. It contains foul, decomposing urine. It is often prevented from emptying by adhesions to other structures. Surgery is indicated.

Such surgical cases may be further subdivided into two groups:

1. Retention diverticulum without vesical neck obstruction.
2. Such with vesical neck obstruction.

In the first group the sepsis can be cleared up well enough by an indwelling urethral catheter to permit removal of the sac. The performance of any suprapubic drainage operation in these cases is to be avoided.

In the second group the sepsis can be overcome but rarely by the use of the urethral catheter to permit a safe operation on the bladder neck, either resection or prostatectomy. Suprapubic drainage is then necessary. The sac can be removed at the first operation, if the patient is fit for such procedure, so that the second operation will be the removal of the obstruction at the bladder neck alone.

There are two chief methods of dealing with the sac itself: 1. Radical excision of the entire sac after inversion. 2. Inversion of the sac within the bladder with removal of the lining mucosa, closure of the orifice, and extra-vesical drainage of the residual shut-off pouch.

The excision of a bladder diverticulum is always an interesting and sometimes a difficult operation. Of course, excision of the diverticulum alone will not be sufficient in the majority of cases. An obstruction should always be suspected until its existence can or cannot be proved.

For the non-surgical cases the best form that palliative treatment can take is to encourage the patient to find some position in which he can perform miction in two parts. Inoperable cases need irrigations of the bladder. The complications are treated according to their nature.

* * * *

Case History

Patient: Mr. L. C. Was seen first on May 28, 1936. Age 31. Barber.

General health fair. Very nervous and irritable. Chronic constipation. Has had usual diseases of childhood with uneventful recovery.

For 12 years patient states he has suffered from bladder trouble, itching and burning sensations during urination, nocturia two to three times, and low back pains, occasionally passing blood and pus. Had a great deal of treatment. At one time was treated for gonorrhea that he did not have with no improvement in his condition.

On cystoscopic examination: A No. 24 Brown Buerger cystoscope was passed with difficulty into the bladder, tightness being noted in the posterior urethra. The bladder contained a great deal of inflammatory exudate. The left ureteral orifice appeared distended and partially obliterated; however, it was easily catheterized with a No. 6 catheter. With Indigo Carmine in the vein each kidney functioned normally. Examination of the specimens from either kidney showed 1-5 w.b.c. from the left kidney, about the same from the right kidney, and a few r.b.c., which were probably due to catheter trauma.

Roentgenologic examination: Flat plate showed the kidneys to be in normal position and no suspicious shadows. A small opening was noted to the left of the left ureteral orifice. A cystogram was made

with 150 c.c. of 12½ per cent sodium iodide solution; showed a moderate sized diverticulum at that point, with no retention.

On September 14, a suprapubic cystotomy was done. The entire sac was pulled into the bladder by suction and removed. The muscular opening of the bladder wall was closed with chromic No. 1 pursestring suture and reinforced with interrupted chromic; the bladder was closed with a hard rubber drainage tube, and the perivesical space drained with cigarette drains. The cigarette drains were removed the fourth post-operative day, and the suprapubic vesical drains removed the seventh post-operative day. He was dismissed from the hospital on the tenth post-operative day; made a most satisfactory recovery. However, he was in about 10 days ago (after three years) complaining of burning on urination, and a cystogram was made in which the antero-posterior position was normal, but lateral visualization showed a small diverticulum posteriorly. He will now come in regularly for dilatations of the urethra to try to bring about an egression of the sac.

* * * *

Dr. Howe—In Closing

Obstruction is generally conceded as the exciting cause. Ninety per cent of all diverticula (in children) are found near the posterior angles of the trigone or in the dome, or vertex, the latter probably coming from an incomplete urachus cyst. With increased intracystic pressure there is trabeculation and the formation of cellules and saccules. Frequently by enlargement of a saccule a diverticulum may develop. The muscular component is usually scant or feeble; because of this a diverticulum is rarely able to empty itself, and, by the maintenance of a pool of stagnant urine, perpetuates infection and predisposes to the formation of stones. CAMPBELL says vesical diverticula are almost always congenital or are immediately secondary to congenital obstruction. In children the sex ratio is 5 to 1, in adults 10 to 1. ENGLISH reported diverticulum complicated by calculus in a boy eight days old. In the babies' hospital a diverticulum was found in a boy seven days old, and HYMAN performed a diverticulectomy on a child nine months of age. OCKERBLAD reports a tumor in a diverticulum, a couple of cases of stone, and one with a ureter opening into a diverticulum. In the latter case the patient complained of pain in the lower quadrant, which was relieved by dilatation of the opening.

A Bug Full of Tricks*

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The above title refers to a despicable little parasite now known as the tubercle bacillus. This microscopic wax covered bit of protein and sugar was plaguing humanity long before the days of recorded history. In subtle, evasive, insinuating fashion, this stealthy enemy of mankind plied its nefarious trade and paradoxical as it may seem, it boldly recorded its own ravages as revealed in exhumed prehistoric skeletons and Egyptian mummies. These angular spines and distorted joints served as stimulating question marks, accentuating the intricate mysteries of a deadly disease, which has followed its host with the ardor of a well-known Biblical character, silently decreeing, "Whither thou goest I will go, and where thou lodgest I will lodge, Thy people shall be my people." So it is unto this day. In spite of all our accumulated knowledge, our destiny is still intimately identified with the tubercle bacillus.

Passing on to the serious study of recorded history, we find that the code of Hammurabi, written more than 2,000 years B.C., indicates a knowledge of tuberculosis. In Deuteronomy, the 15th century B.C., it is said, "The Lord shall smite thee with a consumption and with a fever and with an

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inflammation." In the fifth century B.C., Hippocrates and other Greek writers recognized the essential clinical features of the disease and described them well. They observed its wasting proclivities and appropriately called it "phthisis." Aretaeus, in the second century A.D., gave accurate clinical description of tuberculosis and suggested routine treatment similar to that employed today. Galen accepted the teachings of Hippocrates and recorded his own observations.

In spite of their keen powers of observation and their illuminating clinical descriptions, it is obvious that Hippocrates, Aretaeus and Galen were groping for light while hopelessly submerged in the occult phenomena caused by this baffling bacillus. Apparently Galen never suspected that his own "side stitches" represented a pleuritic manifestation of phthisis. For more than 2,000 years, successive generations suffered and died in the dim light supplied by these renowned Greek physicians. During this long period there was much confusion about the pathological and clinical manifestations of the disease. Scrofula and pleurisy were not recognized as of tuberculous origin and many other extra-pulmonary manifestations passed without identification. Bizarre beliefs and practices befogged the issue, and finally, the many absurd therapeutic measures were climaxed by the royal touch for scrofula, and only occasionally a flickering light appeared on the dim horizon to encourage further investigation.

In 1543, Vesalius published his monumental work on anatomy. In 1546 Fracstorius suggested the modern conception of contagion through microorganisms and expressed the belief that phthisis is due to invisible germs. In 1553 Servetus discovered the lesser circulation; in 1567 Paracelsus wrote about miner's phthisis and boldly broke away from Galenic teachings, becoming one of the first advocates of chemistry in medicine. In 1590, the compound microscope was introduced by Hans and Zacharis Janssen; in 1616 William Harvey announced his discovery of circulation of the blood; in 1650 Franciscus Sylvius declared that tubercles are the cause of phthisis and sensed the connection between scrofula and phthisis. In 1683 Leeuwenhoek described microorganisms and initiated the study of bacteria; in 1689 Richard Morton published a voluminous treatise on consumption, entitled "Phthisiologia." He stressed the teaching of Sylvius that tubercles are connected with and precede phthisis. In 1700, Manget gave the first postmortem report of miliary tuberculosis. About this time Morgagni (1682-1772) was laying the foundation for the study of gross pathology, but refused to perform autopsies on the victims of phthisis because of his fear of contagion.

About the middle of the 18th century, the tuberculosis death rate reached its highest point in statistical columns. This peak of mortality in the midst of universal confusion with reference to clinicopathological manifestations, was coincident with rapidly increasing facilities for the acquisition of knowledge and may have inspired the dawning urge for more specific investigations.

In 1761 Auenbrugger, though unheeded at the time, made an epochal contribution through his *Inventum Novum,* which described immediate percussion. In 1765, William Stark, fresh from the University of Edinburgh, came to London to study pathology under the renowned Dr. John Hunter. He devoted his energy almost entirely to the study of the pathology of phthisis. Contemporaneous with Stark, Robert Whytt described tuberculous meningitis, and Percival Pott first described tuberculous caries of the spine, which justly took his name but remained to be proven of tuberculous origin by Delpech in 1816. Soon thereafter, Marc-Antone Petit described tuberculosis of the larynx and Portal suggested a definite connection between engorgement of lymphatics and pulmonary consumption. William Stark, at the early age of 29, died of miliary tuberculosis as the result of an autopsy wound. Matthew Baillie, having recently graduated in medicine, received an appointment to St. Georges Hospital to work in the autopsy room where Stark had received his fatal infection. No doubt Baillie was inspired by the work of young Stark and sought the opportunity of pursuing the latter's pathological researches under his famous uncle, John Hunter. For 18 years Baillie diligently followed his postmortem studies of diseases of the chest, and made valuable contributions to our knowledge of tuberculosis. He also received an infection of the hand while per-

forming an autopsy but died of advanced pulmonary tuberculosis at the age of 62. His work on "Morbid Anatomy" was published in 1795. His investigations clarified our knowledge of tubercle and differentiated nodular and infiltrating pathological types.

At the turn of the century Robert Willan discussed the relationship of erythema nodosum to tuberculosis, Thomas Beddoes, in his Pneumatic Institute at Clifton, England, was doing interesting work with provocative reports of his investigation, and Benjamin Rush was making valuable contributions in America. Bichat progressed from the organ pathology of Morgagni to tissue pathology within the organ, which was soon to be supplemented by the cellular pathology of Virchow.

After painstaking researches in pathology, Bayle described tuberculosis as a constitutional disease caused by tubercles. He also recognized extra-pulmonary tubercles and gave them proper consideration in connection with phthisis. Flicke says, "Bayle's life and work might be termed a melancholy romance of science. Born in 1774 he died of tuberculosis, probably contracted at the autopsy table, in 1816 at the age of 42." What he accomplished in a short time brought the knowledge of tuberculosis further forward than all that had been done before him.

In a volume on *Pulmonary Phthisis*, he carefully presents the results of his clinical observations, checked by autopsies on more than 900 individuals dying of consumption. His exhaustive studies at the bedside and in the dead room enabled him to have a better understanding of tuberculosis in all its stages and varied pathological phases, than any of his predecessors. He divided the disease into four stages: (1) occult; (2) incipient; (3) confirmed, and (4) advanced. He exhibited advanced knowledge of pleurisy and pleural adhesions but did not understand the relationship as we know it today.

Early in the 19th century, Covisart translated Auenbrugger's *Inventum Novum* and popularized percussion, which in spite of a previous French translation, had never achieved general recognition. Strange to say, Covisart first learned of percussion, not through the French translation of 1770, but through the influence of Maximilian Stoll, who had succeeded de Haen as director of the Clinic in the Allgemeines Krankenhans, University of Vienna. In addition to his wide influence in the field of clinical investigation, Stoll manifested a special interest in tuberculosis, perhaps because of its ravages in his own body to which he succumbed at the early age of 46. Covisart's greatness is attested by the following quotation from his brief preface to the translation of Auenbrugger's pamphlet.

"I know well how little reputation nearly all translators and most commentators earn and hence I might have secured for myself an authorship if I had published a work on percussion based upon a recasting of the writings of Auenbrugger. I then would have sacrificed the name of Auenbrugger to my own vanity; that I did not wish to do; I wanted to snatch him and his beautiful, regularly made discovery which he with entire propriety called a new invention, from forgetfulness."

Later Piorry introduced the ivory pleximeter which was soon to be replaced by the more satisfactory use of the fingers. Piorry's mediate percussion, combined with Laennec's new methods of auscultation, exerted a profound influence upon the progress of physical diagnosis.

We now come to the significant awakening with reference to clinical and pathological investigations with Portal, Stark, Baillie, Bichat, Bayle and Covisart supplying a stimulating background for the unprecedented work of Laennec and Louis in the first quarter of the 19th century. Laennec promptly grasped the significance of the clinicopathological implications and with an avid genius, he appropriated all previous scientific advances as he entered upon his monumental career. In spite of the handicap of progressive tuberculosis in his own person, he worked with remarkable industry and conspicuous discrimination. Before he was 22 years of age he had drawn up a minute history of nearly 400 cases of disease, which served as a foundation for his future researches and discoveries. When Laennec came upon the scene, the diagnosis of diseases of the lungs and heart was still more difficult than that of other internal organs. In a short time he had made the most exacting diagnostic tasks relatively easy. In

half the time now allotted for a medical education, virtually without chart or compass, he "Observed, recorded, tabulated and communicated" practically all that is now taught with reference to the physical diagnosis of diseases of the thorax. His voluminous work of nearly 800 pages on auscultation and diseases of the chest was published in 1819 and translated into English by Forbes in 1821.

After giving full credit for Covisart's revival of Auenbrugger's neglected invention, it is doubtful whether the remarkable advances of the 19th century would have followed each other so rapidly without the stimulus of another famous invention, the stethoscope. Immediate auscultation in haphazard fashion had been practiced since the days of Hippocrates, but it remained for Laennec, through his ingenious invention, to perfect this method of examination and to extend and record its diagnostic possibilities. Quoting from a recent article by Korns, "The great merit of Laennec's invention lay not so much in the instrument itself, as in the fact that it served to focus the attention of the entire medical world on the auscultatory method. Rolleston, in his Harveian oration (1928), expresses this idea as follows: "though the stethoscope has some obvious advantages over the naked ear, the enormous advances that followed its introduction were not so much due to the stethoscope as a mechanical instrument, as to the psychological effect that this new method exerted on Laennec, who otherwise would not have so ardently pursued auscultation as a means of diagnosis."

At the age of 45, on August 13, 1826, Laennec died of advanced pulmonary tuberculosis with associated tuberculous enteritis. In keeping with his genius for independent accomplishment, two hours before his death he removed the rings from his fingers in order to save others the trouble.

The great clinician, Louis, who was contemporary with Laennec and survived him by many years, also suffered from tuberculosis, but lived to be 85 years of age. Louis made use of the facts Bayle and Laennec had brought to light and gave them added significance through his own observations, his writings and his wonderful influence as a teacher. He carefully correlated the symptoms and the pathology of tuberculosis; he continued and amplified the study of clinical manifestations in the light of autopsy findings. Finally, when 34 years of age, he gave up general practice in order to more satisfactorily pursue his scientific investigations. Louis established the numerical or statistical method of investigation in medicine. Through his teachings and his pupils, he was largely responsible for the establishment of clinical medicine, with a special interest in physical examination, throughout the world. The results of his studies were published in a volume of 600 pages under the title, *Louis' Researches on Phthisis.* His influence is evident wherever the principles of clinical medicine are taught. In the United States, his teachings were perpetuated through such pupils as James Jackson, Jr., Oliver Wendell Holmes, George C. Shattuck, William Pepper, Gerhard, Stille, Power, Swett and Clark.

Contemporary with Louis, Virchow, advancing from Bichat's tissue pathology to cellular pathology, made valuable contributions to our knowledge of tuberculosis. However, he joined the dualist Schonlein, who coined the word "tuberculosis," in order, as he thought, to differentiate the nodular tubercles from the cheesy patch characterizing phthisis. Thus is seen the perspicacity of Laennec with reference to the "unity of phthisis" blocked by the unfortunate and misguided application of a scientific advance. Not until the discovery of the tubercle bacillus were the dualists routed and the unity of tuberculosis finally established.

In the University of Vienna the eccentric, impetuous Skoda (1805-1881), taking his cue from the French school, enthusiastically pursued the study of percussion and auscultation, with emphasis upon the investigation of tonal or sonorous phenomena. Skoda's skill in diagnosis so inspired Turkheim, minister of medical education, he established a special department on chest diseases with this great clinician in charge. Skoda's appointment as professor of medicine in 1846 and his skill in physical diagnosis attracted students from all parts of the world. However, the magnetic attraction of the University of Vienna for students of clinical medicine was shared by an illustrious contemporary of Skoda. The rapid rise of this school to the highest rank was largely due to Ro-

kitansky (1804-1878), the world's greatest clinical pathologist. Skoda's work was brilliantly complimented by the latter's illuminating demonstrations in the dead room. Rokitansky handled 1,500 to 1,800 autopsies annually. It is said that he performed over 30,000 postmortem examinations during his lifetime.

By the middle of the 19th century the stage was set for a great scientific awakening. In 1865 Villemin demonstrated the specific nature of tuberculosis through inoculation. Five years later, Gerlach proved that milk from tuberculous cows conveyed the disease, and in 1873, Edwin Klebs, through feeding experiments, produced bovine tuberculosis. Medicine was moving into a new era. The consolidated methods of clinical, anatomical and pathological studies were soon to be supplemented by modern laboratory methods.

It seems worthy of note that much of the progress in physical diagnosis and pathology has been evolved through a sustained desire to solve the mysteries of tuberculosis. It is equally interesting to note that a large percentage of the most zealous investigators have suffered from tuberculosis; some apparently experiehcing an excitation of genius which spurred them on to greater accomplishments, thus partially compensating for an early surrender to the enemy they so ardently pursued. It seems that the tubercle bacillus, in spite of its magical powers of evasion and deception, was preparing the way for its ultimate discovery and the gradual surrender of many of its secrets.

In 1856 Virchow, in the city of Berlin, established the first pathological laboratory. It served as a model for nearly all laboratories in Germany and in other countries. The first hygienic laboratory was opened by Pettenkofer in 1878. Robert Koch, stimulated by the discoveries of Pasteur, had already begun his epochal researches. Allen K. Krause, discussing Robert Koch's rapid and uninterrupted march from obscurity to universal fame, says: "Just before Koch emerged from Posen, Cohn, in writing his great work on botany, had scrapped what was then known about bacteria into the nondescript genus of *chaos*: when, in almost a trice and out of a most unexpected source, was born a new science, *bacteriology*. Stone by stone the builder raised his structure, year by year the new science took on form and fullness: from the cause and nature of anthrax in 1876; to the smear-preparation and staining methods of '77; the parasitic excitants of wound infection in '78; and then the crowning and astonishing plate-isolation method to signalize his assumption of the reins on *Luisenstrasse* in 1881. Already had been forged the tools to lay bare the gonococcus (Neisser, 1879), the pneumococcus (Sternberg, 1880), the typhoid bacillus (Eberth, 1880), the glanders bacillus (Loeffler, 1882)—bacteria of disease, as Koch put it, falling into his and his pupils' laps "like ripe apples from a tree." On his first trip to Breslau he had met Weigert, Cohnheim's associate, and the former's young cousin, Paul Ehrlich; and Ehrlich was doing a little more than *play* with aniline dyes, whose possibilities had long intrigued his pathologist-cousin Weigert—he was making thin smears of blood on cover-slips and staining these to study the cells.

So the stage was set for progression from Weigert to Ehrlich to Koch; and we follow the chain from Weigert's methylene-blue and Ehrlich's thin heat-fixed smears to Koch's stained smear-preparation, to demonstrate those early pathogenic bacteria. Gelatine, solid and transparent, was the culture medium, streaked with the material to be studied. And until 1882 these simple methods, fortified now by the plate-isolation process, promised to disclose the causes of all infectious diseases.

Then Koch struck a snag.

He began the search for the cause of tuberculosis in the year 1881. He got busy on known tuberculous tissues with his trusty ordinary methylene-blue, and saw—nothing; that is, nothing that might be imagined to be an adventitious product—a germ; no round dots as in wound infections and gonorrhoea, nor rod-like particles as in typhoid fever and anthrax. But, supreme adepter and borrower that he always was, he soon had recourse again to a Weigert tissue-stain method: he "fortifies" his methylene-blue stain with potash, dips in the mixture a fresh section of tissue, and now, first of mankind, beholds within the blue background of tissue elements the fine blue streaks that by this time had come to mean "germs." Thus ended step number one, the demonstration of something extrinsic to tissue and its degrada-

tion products, the presumption that Koch was gazing at the physical cause of consumption. The perfection of this first step, the refinement of the method by adding a counterstain, Bismarch-brown, was a mere detail to be worked out presently."

There were other snags but the story of Koch's "step-by-step proof of the specificity of the tubercle bacillus" is so well known it is not necessary to repeat it here. It is important to note that the snags encountered were due to the fact that the tubercle bacillus is a non-conformist and only a Robert Koch could have successfully accomplished the task. Again quoting Krause: "Given the same circumstances, the same pioneer quest, the same limited knowledge of possibilities, the same imperfection of initial media—and the view is almost compelling that only the rarest of investigators would have maintained the prolonged and heartbreaking vigil for the earliest "showing through" of minutest particles that belonged, as yet, only to the shadowy realm of "working hypothesis."

This perfect performance which marked the zenith of Koch's career and its presentation in irrefutable terms, silenced the dualists and forever established Laennec's claims for the unity of tuberculosis. Even Virchow, still supporting the dualists and ever ready for controversy, quick of mind and body, remained speechless and immobile, thus accentuating the profound silence that followed Koch's dramatic presentation of the world's most perfect bit of research. Something new to mortal ken had sounded the death knell of dualism, and to the war horse of pathology, it was like the passing of a cherished child. Though, on this occasion, he left the Berlin Physiological Society without a word of response, it is worthy of note that in the language of Krause, "When, full of years and every honor that a man of medicine can accumulate, he passed on to eternal peace, he could not reconcile himself to the idea of the all-embracing and unified cause of that appalling diversity of effect that tuberculosis comprehends."

The discovery of the tubercle bacillus in 1882 did not satisfy Robert Koch. His dauntless spirit was soon busily engaged in search of a cure for tuberculosis. In 1890, his announcement of tuberculin caused a furor throughout the medical world.

Koch, having discovered the specific cause of tuberculosis, and, as he first thought, having found a cure in tuberculin, it then seemed reasonable to anticipate the end of humanity's greatest scourge. Unfortunately, we were doomed to descend from this hopeful height, but the settling point left us far above the level attained by Laennec and Louis. Koch's discoveries led to many valuable epidemiological, diagnostic, therapeutic and preventive principles, and initiated a sustained search for additional knowledge which has reached an astounding magnitude.

Though disappointing as a cure, tuberculin proved to be a valuable diagnostic agent and helped to stress the ubiquitous presence of the tubercle bacillus. Through Roentgen's discovery of the X-ray, the value of inspection was immeasurably increased. Later the bronchoscope extended our scope of vision and revealed pathological manifestations of tuberculosis not previously suspected.

Time will not permit a detailed discussion of therapy, but a brief reference to its development with its present sad limitations, is sufficient to further emphasize the baffling problems of the tubercle bacillus and its protean manifestations.

Immediately prior to Koch's epochal discoveries, Boddington, Brehmer, Dettweiler and Trudeau pioneered in sanatorium management, which has been so successfully pursued with untold benefit to patients, a wealth of accumulated clinical, pathological and therapeutic knowledge, and statistical data difficult to estimate. Collapse therapy, with its multiple surgical possibilities, is now a matter of common knowledge.

In spite of all that has been accomplished, the unsolved problems in the field of tuberculosis are infinite and in the words of David Riesman, "No matter what we take away from infinity, infinity remains."

To briefly refer to the problems now in the scope of our consciousness, to say nothing of those not yet conceived, would require a large volume. To merely enumerate the research projects, now devoted to these problems, would fill another great volume. The bibliography on the tubercle bacillus and tuberculosis, as reflected in

the quarterly Index Medicus, now occupies 25 times the space devoted to these subjects 30 years ago. The space devoted to tuberculosis is 12 times that allotted to the, now popular, subject of pneumonia, and four times that given to syphilis.

This voluminous literature on tuberculosis indicates that constantly we are making new observations of age-old conditions in this infinite field which still intrigues all investigative minds.

Even though our command over the course of natural phenomena has speeded up immeasurably since Harvey urged us to travel in the path which "nature walks," we are unable to find our way to a full understanding of the tubercle bacillus. With all our studies of immunity and infection; the chemical fractional analysis of the tubercle bacillus; the cultural mutations with their varied morphological forms; our modern advances in diagnosis and therapy; tuberculosis still goes where we go, lodges where we lodge, and remains the greatest killer during the most useful period in the cycle of life.

Perforation of Hollow Viscus*

Roentgen Aspects

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The first single case report of the value of X-ray in the diagnosis of perforation of stomach and intestine was presented in 1915. The first statistical series of cases on the frequency of spontaneous pneumoperitoneum occasioned by peptic ulcer were published by Singer and Vaughan in 1925 and another more complete series in 1929[1]. These authors stated then, "we know of no single symptom or sign in perforated peptic ulcer which is so constant or so free from possible misinterpretation as spontaneous pneumo-peritoneum."

The frequency of air under the diaphragm in perforated peptic ulcers, as reported by various authors, varies from 60 to 90 per cent. In Singer's and Vaughan's series of over 80 cases, 85 per cent of all their cases showed roentgen evidence of perforation, verified by operation or postmortem. A more recent series, reported by Dr. S. E. Johnson[4] of Louisville City Hospital of Kentucky, showed 83 per cent of his cases of proven perforated peptic ulcer to have been diagnosed by X-ray. Dr. G. Petren[7] of the Surgical Clinic of Lund, Germany, found but two-thirds of his proven perforated cases to show free air in the peritoneal cavity.

We know that no other abdominal catastrophe requires a more prompt diagnosis and treatment than does intestinal perforation. The surgeon today recognizes the value and is often guided by the roentgen findings in intestinal perforation. Since the recognition of a spontaneous pneumo-peritoneum is so pathognomonic of perforation, why are we not able to demonstrate this pneumo-peritoneum in over 90 per cent of the perforated intestinal cases submitted to X-ray examination? Is it due to improper technique or insufficient study? Is it due to the fact that the lesion is walled off by adhesions at the time of rupture? Is spasm a factor? Is it due to the size or location of the perforation? To the interval of time between the perforation and time of X-ray?

An attempt to answer these questions prompted me to write this paper.

A few months ago, a middle aged salesman entered the hospital in the typical jack-knife posture, four hours after his perforation. At *no time* since the perforation did he or could he lie down. The X-ray studies were made as soon as the patient entered the hospital, in the sitting-up position. No air under the diaphragm was visualized. A large amount of air was present in the magenblasse of the stomach.

*Read before the Section on Dermatology and Radiology, Annual Meeting, Oklahoma State Medical Association, Oklahoma City, May 2, 1939.

At operation, a perforation eight mm. in diameter was found on the lesser curvature side of the pars pylorica. No adhesions of the omentum or liver, no exudate or fibrin was seen over the site of perforation. I therefore assumed that the failure to prove the presence of the perforation by X-ray in this case was due to my failure to place this patient on his left side in supine position, before I did the X-ray examination. The large amount of air in the stomach was trapped in the cardia, and never had the chance to get to the site of the perforation, which would have been the case if he had assumed the above mentioned position.

I thought I had stumbled on an additional aid in roentgen diagnosis of peptic ulcer perforations. On review of the literature, I found that Singer and Vaughan made this same observation 10 years before. When their original examination on admission failed to prove the perforation, these authors were able to demonstrate free air in the peritoneum in several of their cases, after the patient was placed in the left supine position for a half hour to several hours.

Proper X-ray techique will therefore undoubtedly increase the percentage of cases of peptic ulcer perforations that are roentgen positive. But there are several other factors in 10 to 15 per cent of cases that prevent the leakage of air through the perforation, as:

1. *Spontaneous spasm* at the site of perforation. The importance of this factor was demonstrated by Dr. Poas[9]. He produced in dogs an artificial tear in the anterior duodenal wall close to the pylorus. The pyloric spasm that was produced persisted for several hours and prevented the passage of gastric contents into the duodenum and thus into the peritoneal cavity.
2. Deposition of fibrin over the perforation, adhesions of omentum or neighboring organs to site of ulcer, and impacted mucous plugs are other causes for spontaneous closure of the perforation soon after the rupture.

   A common explanation that I have heard radiologists offer to referring physicians when the radiologist could not demonstrate the perforation by X-ray was that the lesion was probably a walled-off penetrating ulcer that had perforated. At the operating table, this type of walled-off perforation is rare, less than two per cent of all cases.
3. The size of the perforation—The smaller the hole, the more likely the possibility of a spontaneous closure before the air has had a chance to get through into the peritoneum.
4. The site of the perforation—In about 121 cases reported by the Russian Sosnyakov[5] of Leningrad, the perforations were localized in all their cases on the anterior wall of the stomach and duodenum; only in one case was the perforation found on greater curvature side and in only five cases near the cardia of the stomach. Since 90 to 95 per cent of all ulcer perforations occur at the pylorus and duodenum, the procedure of placing the patient in left supine position for a short period of time prior to X-ray study, will undoubtedly increase the number of roentgen-positive cases.

   The clinician does not usually need the X-ray to diagnose a perforation of a posterior wall duodenal ulcer. The marked gastric hemorrhage and melena are characteristic. The site and nature of other peptic ulcer perforations are determined by means of barium meal X-ray study.
5. The interval of time between the perforation and X-ray study — In most cases, this is not a definite factor in the roentgen diagnosis. Most cases will show free air in the peritoneum soon after the perforation. Placing the patient in the left supine position for a half to several hours after the first roentgen examination is negative, will reveal in many cases a *delayed pneumo-peritoneum*, i.e., in small perforations where the spasm is relieved or the plug dislodged.

Technique

1. Sitting up position, posterior-anterior view. The patient should sit up for a few minutes before the film is taken in order to permit the free air in the peritoneum to ascend to the diaphragm. The voltage and time of X-ray exposure should be slightly more than is required for a chest film.

2. Lateral position—The patient lies on his left side, right side up, film placed along lower right ribs, X-rays directed perpendicular to the body.
3. The majority of perforations will be demonstrated by the above positions. If the X-ray findings are negative by above, then the left supine position for one to several hours, and X-raying at intervals, as advocated by Singer is recommended. The X-ray studies that I have made reveal that the left supine *Trendelenberg* position will permit a much larger amount of air to collect in the pars pylorica. In the straight left supine position, X-ray studies by means of barium meal reveal most of the air to be collected in the esophageal end of the stomach and only a small amount on the lesser curvature side of the pars pylorica.

   If there is no air in the stomach at the time of perforation, no air of course could go into the peritoneum. When X-ray studies show such an absence of air in the magenblasse, I have my patients swallow some air, and then use the above technique.
4. Fluoroscopy of the patient while on the stretcher in sitting-up position should supplement the film studies. In several of my cases, the fluoroscopic study revealed a small sub-diaphragmatic collection of air that was not observed in the films. In many cases, the thin air collection was seen in expiration and not in inspiration. Often the oblique view shows the air collection best. Again, the free air is often hidden behind the air in the cardia.

   The fluoroscopic study is of value in differentiating the free collection of air in the peritoneum from air within the colon or stomach, from emphysematous air pockets at base of the lungs, etc.

Clinical Types of Peptic Ulcer Perforation

Some of you have encountered surgeons who claim they never require roentgen aid for a diagnosis of intestinal perforation. These surgeons apparently have seen but few cases, and are familiar only with the classical text-book picture of perforation. It is therefore essential that the surgeon and radiologist consider and recognize the clinical types of peptic ulcer perforation:

1. Classical type — The patient experiences sudden excruciating pain in the abdomen, causing him to assume the jack-knife *sitting-up* posture. The rigidity and abdominal tenderness is not equaled by any other abdominal catastrophe. The patient's attitude and symptoms remain unabated until operation.
2. Singer and Vaughan in 1933, designated this second type as the "formes fruste" case. The patient experiences the same sudden knife-like abdominal pain at the time of perforation. But within one to several hours, the patient's symptoms have abated and he is often ready to discharge the surgeon. Mild to moderate rigidity and tenderness are the only clinical findings at this time. In this type of case, the roentgen demonstration of a spontaneous pneumo-peritoneum achieves its greatest diagnostic value.
3. The explanation for the change of symptoms in "forme fruste" cases is that the perforation has spontaneously closed. Under judicious care as by continuous gastric siphonage and parenteral feeding the perforation stays closed without operation. But if the above condition is not recognized and the patient overloads his stomach, the perforation again becomes patent with a repetition of acute symptoms. A third and even a fourth similar episode may occur, before the classical picture of perforation occurs. Singer and Vaughan first described this type of case in A.M.A. in 1934, under the title of *"Intermittent Leakage From Perforation of Peptic Ulcer."*[3] I refer you to their paper for further details.
4. The fourth type — The perforating peptic ulcer simulating the gastric crises of Tabes, is described in a recent number of Staff Meetings of Mayo Clinic[5]. This type of ulcer syndrome is caused by deep penetration of the ulcer with invasion of tissues contiguous to stomach and intestines. This type is, of course, rare. Diagnosis is proven by X-ray studies by means of barium meal.

   Other types of clinical pictures oc-

cur, being often confused and therefore must be differentiated from (1) perforated gall bladder, (2) pancreatitis, (3) acute coronary occlusion, (4) mesenteric obstruction, and (5) intestinal obstruction.

Spontaneous Pneumoperitoneum due to perforations other than peptic ulcers:

1. Perforations of colon and stomach after gunshot wounds or other missiles. A perforation of the small bowel by a foreign object does not produce pneumoperitoneum, since the small bowel normally does not contain air.
2. Perforated carcimona of stomach or colon.
3. Leakage from enterostomy, colostomy, and anastomosis, etc.
4. Typhoid or tuberculous perforation of small bowel, perforation of appendix (occasionally).
5. Perforation from diverticulae or polypae of the large bowel or duodenum.
6. Perforation of the uterus in abortions.

Spontaneous Pneumoperitoneum occurs in other conditions other than perforations, as:

1. Following recent operations or trauma to abdominal wall by knife, gun, etc.
2. Abdominal paracentesis.
3. Tubal insufflation in Rubin test.
4. Previously induced pneumoperitoneum.

Pneumoperitoneum must be differentiated from gas-containing organs around the diaphragm, as:

1. Hepato-diaphragmatic interposition of the large intestines. A good series of such cases was recently reported by Dr. Kolju[6].
2. Mobile pylorus of the stomach and mobile colon that intermittently assume a sub-diaphragmatic position. This type of case was recently reported by Dr. Singleton of Toronto.
3. Sub-phrenic abscess, characterized by small transparent area within an area of increased density. This collection of air between the liver and diaphragm is fixed, whereas the air in perforation changes its position with change of patient.

(Lantern Slide Demonstration)

Conclusions

1. Four major clinical types of peptic ulcer perforation should be recognized —the classical type, forme fruste, intermittent leakage type, and gastric crisis type. X-ray is of greatest diagnostic value in second and third type.
2. The factors responsible for closure of the perforation or failure to show a spontaneous pneumoperitoneum were discussed. But with proper X-ray technique, at least 85 per cent of all cases of peptic ulcer perforation can be diagnosed by X-ray. The X-ray technique is described.
3. Air containing viscus under the diaphragm and various types of recently induced pneumoperitoneum are the roentgen findings to be differentiated before making a positive roentgen diagnosis of intestinal perforation.

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O.U. Medical Alumni Directory

The University of Oklahoma Association has started preparation of a Medical Alumni Directory to be published as the August issue of Sooner Magazine, the monthly publication of the alumni association.

The directory will contain alphabetical and geographical lists of all graduates of the University of Oklahoma Medical School, and also will contain names of former O.U. students who obtained medical degrees elsewhere and are now in the medical profession.

All alumni of the medical school and former students now in the profession, are urged to report their correct addresses to the Alumni Office, University of Oklahoma, so that they will be properly listed in the directory.

Copies will be sent to all members of the University of Oklahoma Association, and a few extra copies will be printed for sale to others desiring the directory. Orders for individual copies must be placed in advance, however, and accompanied by payment of $1.00 per copy.

Perinephritic Abscess*

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The records of 33 patients with perinephritic abscess were studied. In 30 cases the diagnosis was corroborated by surgical findings, while in three, the pathology was disclosed at autopsy. The group presented themselves at the University Hospital during a period of 17 years. In this interval 95,000 hospital admissions were recorded.

The anatomical peculiarities of the perirenal structures predispose the presence of pathological variations. The kidney is surrounded by a pale layer of fatty tissue, more abundant posteriorly and inferiorly. Continuous with a similar layer about the ureter, this fatty tissue extends into the pelvis and is circumscribed by the renal and ureteral fascia. The possibility of extension of infection within the periureteral fascia must be emphasized. Rolnick[1] demonstrated such ease of dessimination by injections of radiopaque medium within the periureteral fascia of postmortem specimens. Downward extension followed injections near the renal pelvis. An upward dessemination occurred along the ureter when injections were made at the base of the bladder. A somewhat similar process may have occurred in the patients herewith described:

Case No. 88954, Mrs. E. M., white, age 32. On the seventh postoperative day following a left salpingo-oophorectomy and appendectomy, the patient suffered a rather rapid onset of severe pain in the right posterior lung base. The temperature and pulse, which had been normal, rose to 101.6° and 120 respectively. Thereafter daily temperature elevation was 101°, and pulse rise 120. The respirations remained normal. Five days after onset a mass was palpated in the right costo-iliac region. Four urinalyses showed a moderate number of white blood cells. The leucocytes were 13,000 with neutrophiles 86 per cent. Eight days after the onset an incision and drainage of the right perirenal space was performed. There was an extensive abscess at the inferior pole of the right kidney with indications of pelvic connection. Postoperative recovery was uneventful.

Case No. 79,107, Master J. M. W., white, 19 months of age. There had been pain in the left hip of one month duration, fever, and inability to bear weight on the left lower extremity. A gradual flexion deformity of the left thigh on the abdomen had developed. An outpatient diagnosis of infectious arthritis of the left hip was made and admission requested. After admission, four days later, careful examination disclosed a mass in the left subcostal and costo-iliac regions. There was tenderness and muscle spasm in the left costovertebral angle and evidence of psoas spasm. There were daily elevations of temperature to 103.6°, of pulse to 128 and of respiration 28. Urinalysis showed one plus albumin. The leucocytes were 17,000 with neutrophiles 86 per cent. X-rays of the dorsal and lumbar spine were negative. Incision and drainage of the left perirenal space disclosed an abscess containing 100 c.c. of thin purulent material and located inferior to the kidney. Recovery was uneventful.

The first case presents the possibility of an upward dessimination while the latter, because of downward extension caused the clinician to attribute the pathology to the hip joint. The renal fascia is closely attached to the kidney pedicle, thus preventing any transverse spread of an infection.

*Read before the Section on Genito-Urinary Diseases and Syphilology, Annual Meeting, Oklahoma State Medical Association, Oklahoma City, May 3, 1939.

The retrorenal portion is more commonly known as the fascia of Gerota. Posterior to the fascia of Gerota is a second fatty layer situated in close proximity to the muscles and fascia of the posterior abdominal wall.

The blood supply of the perinephrium is derived from branches of the main renals, the adrenals, spermatics, and the first lumbar, combining to form a vascular network about the kidney. The intercolumnar arteries of the renal cortex after penetrating the capsule contribute to this vascular network. Hence the perirenal fatty layer may be involved by a blood borne infection from suppuration existing at any place in the body and without primary kidney pathtology.

The lymphatics of the perinephrium communicate freely with the subcapsular kidney group and drain into the upper lateral lumbar nodes.

Perirenal suppuration may ensue from three sources: an extrarenal direct blood borne infection, rupture of single or multiple cortical abscesses, and secondary to severe kidney infections. The latter may transpire by blood or lymphatic anastomoses or direct extension. An abscess of extrarenal direct blood borne origin as from foci of infection is most likely to be diffuse, unilateral and fairly extensive. It is usually situated posterior to the kidney, at the mid and inferior portions. The fatty tissue at the superior pole is of small amount and not often involved by a blood borne infection.

Secondary extension from a cortical abscess may occur at any point in the perinephrium. The involvement is frequently small and resolution often results from conservative management. Perinephric abscess arising from a pyelonephritis is fortunately less frequent. Predominant kidney pathology masks the diagnosis of the complication. Three perinephric abscesses found at autopsy were secondary to a severe pyelonephritis. Obstruction at some point in the urinary tract contributed to the kidney infection. One patient had a large hypertrophied prostate, the second an impassable urethral stricture and the third, incarceration of the ureter by a parametrial extension of a carcinomatous cervix.

The more extensive perirenal abscesses usually rupture the fascia of Gerota and underlie the fascia and muscles of the posterior abdominal wall. Occasionally pathological processes of nearby organs secondarily involve the peri and pararenal tissues. Important sources of such involvement are perforated ulcers of the posterior duodenal wall, a neglected retrocecal, retroperitoneal appendicitis, and a ruptured liver abscess of the dome with subphrenic spread and secondary extension.

In the direct blood borne extrarenal infection of the perinephrium, the most common primary foci of suppuration are furuncles, carbuncles, paronychia and less often osteomyelitis and gonorrhea. Upper respiratory infections, scarlet fever, influenza and other general infections contribute to the etiology. Fifteen of 30 patients had a history or physical findings of a previous or existing infection as a possible primary foci. The history was questionable in five and not listed in eight records. In six patients trauma seemed to play an important role. Two were injured in the respective costo-iliac areas during wrestling matches. One was thrown from a horse striking the side. Another delivered ice for his livelihood, with symptoms on the side that the ice was carried. The fifth was kicked in the kidney region during a fight and the sixth strained the respective costovertebral area during lifting.

Hemorrhage into the renal retroperitoneal area from trauma predisposes to bacterial invasion and in this event may result in a traumatic abscess.

TABLE I

Etiological factors contributing either singly or in combination to the development of a perinephritic abscess.

Types of Infection	Number of Patients
Furuncles	5
Influenza	5
Questionable	5
Gonorrhea	2
Measles	1
Typhoid	1
Known direct extension	3
Trauma	
Strains	4
Direct injuries	2
Postoperative	1

Twenty-one of the 33 patients were between the ages of 20 and 40. The youngest was 19 months and the oldest 63 years of age.

TABLE II

Distribution of patients in age groups.

Decades	Number of Patients
0-10	2
10-20	5
20-30	13
30-40	8
40-50	3
50-60	1
60-70	1

Twenty-six patients were of male and seven of the female sex. One was of the colored race. One patient had bilateral involvement, secondary to bilateral pyelonephritis. Thirty-two presented a unilateral abscess, 21 on the right and 11 on the left.

The symptomatology of the perinephric abscess in its early stages is confusing. Eleven of the 26 had had symptoms for four weeks or longer prior to hospitalization, and one for five months.

TABLE III

Duration of symptoms before hospitalization.

Duration	Number of Patients
1-7 days	4
7-14 days	2
14-21 days	9
21-28 days	3
28-35 days	2
35-42 days	2
42-49 days	2
8 weeks	1
5 months	1

It was necessary to keep some patients under observation in the hospital for a considerable period. In 18 of 28 the observation extended beyond three weeks, and in two as long as two months.

TABLE IV

Hospital days before surgery.

Days	Number of Patients
1-7	1
7-14	2
14-21	7
21-28	5
28-35	7
35-42	
42-49	4
49-56	2

At onset, the majority of patients will complain of pain in the corresponding costovertebral angle, more in the nature of a dull ache. Twenty-three of 33 recalled low grade pain as the first symptom. Seven had abdominal and one hip joint pain. Chills and fever were the first symptoms in one and in another, urinary retention. Usually the onset in those cases not attributed to trauma is gradual with a continuous persistent fever. In the six patients with a history of trauma, the symptoms gradually subsided for five to ten days. Pain and tenderness then reoccurred associated with evidence of infection. The presence of abdominal pain may be misleading. The pathology is often attributed to the gall bladder, appendix or other organs. In an abscess about the lower pole of the kidney, the symptoms may resemble those of a retrocecal appendicitis. As an example of such a diagnostic problem:

Case No. 70166, Mr. J. C., white, age 22. Three weeks prior to admission an aching pain was noted in the right hip aggravated by movement and riding in an automobile. Gonorrhea developed one week before admission and treatment was received. The patient had remained constantly in bed. There had been a persistent diarrhea for three days. More relief was noted with both thighs flexed on the abdomen. Admission temperature was 102.4°, pulse 138, and respiration 24. The patient was toxic and dehydrated. Tenderness was present in the right lower quadrant, less pronounced in the right upper quadrant and marked in the right costovertebral angle. Rigidity presented in the right lower quadrant extending into the right costoiliac region. Repeated urinalyses were negative. The blood leucocytes were 18,100 with polymorphonuclears 93 per cent. A retrocecal appendicitis was feared; appendectomy was performed with a McBurney incision. The appendix was retrocecal and only mildly injected. The blood leucocytes dropped to 9,000; however, there was a daily postoperative fever to 101° or 102°. On the eleventh postoperative day pain increased in the right costovertebral angle and radiated to the epigastrium. Diarrhea continued and tenderness increased in the right costovertebral angle. Rigidity became quite marked at this point. Blood leucocytes were 26,200 with 90 per cent neutrophiles. Five postoperative urinalyses were negative except for a few red blood cells in one specimen. Sixteen days after hospitalization an incision and drainage of the right costovertebral angle disclosed a perinephric abscess.

A past history of urinary symptoms was given by six patients. At admission 13 complained of dysuria, frequency or nocturia. Dysuria was the sole urinary symptom in nine. Eleven complained of nausea and vomiting and seven had had chills. One had pronounced night sweats and another a persistent diarrhea.

In the typical case of perinephritic abscess, the physical findings will vary with the duration of the disease and the location of the process. Authors agree that occasionally two months or longer may transpire before the correct diagnosis is apparent. In other instances the physical findings will be characteristic within a few days. This occurred in the following case:

Case No. 94162, Mr. R. B., white, age 17. Symptoms had been present for six weeks but more pronounced during the last three weeks. Paroxysms of severe sharp pain would occur in the right lumbar region, more severe at night and of variable duration. The pain often radiated to the right inguinal region and upper inner thigh. The attacks were followed by slight dysuria and frequency. The admission temperature was 100.6, pulse 88 and respiration 20. Complete physical examination was negative except for slight tenderness to percussion in the right costovertebral angle. Ureteral calculus, right, was thought to be present. Seven urinalyses were negative except for red blood cells and white blood cells in two specimens. Blood chemistry, hemaglobin and red blood count were normal.

The Wasserman and malarial smear were negative. The white blood count was 12,900 with polymorphonuclears 90 per cent. Cystoscopic, ureteral catheterization with urine cultures and retrograde pyelograms were negative. Eight days after admission the pain and tenderness increased in the right costovertebral angle. Direct questioning elicited a history of furunculosis during the last three months. Examination showed evidence of a right psoas spasm with pain to hyperextension of the right hip. The patient was more comfortable with the right hip flexed. There was fullness of the right costovertebral angle and scoliosis of the spine with convexity to the left. Increased abdominal reflexes on the right and some hypersensitiveness of the right first lumbar nerve distribution were found. Perinephritic abscess, right, was suspected and incision and drainage in the right lumbar region corroborated the diagnosis.

A definite conclusion after one examination is not often possible. Repeated examinations are usually necessary in both horizontal and upright positions. Only 12 of the 30 operative cases were diagnosed correctly at admission.

TABLE V

Impression received by the clinician after one examination.

Impression	Number of Patients
Perinephritis Abscess	12
Pyelitis	6
Psoas Abscess	3
Pyelonephritis	2
Ureteral Calculus	2
Acute Appendicitis	2
Postoperative Pleurisy	1
Infectious Arthritis of Hip	1
Renal Calculus	1

Physical findings will include scoliosis of the spine with convexity toward the opposite side, spasm of the corresponding erector spinae muscle group, marked tenderness to percussion in the related costovertebral angle and palpable or percussion tenderness extending into the costo-iliac region with variable spasm of the underlying muscles. The patient seeks to keep the hip joint flexed and pain is experienced to extension and hyperextension. There will be a variable degree of voluntary rigidity of the paraumbilical and mesial anterior costo-iliac region. A mass may be palpated fading into the costo-iliac area. If investigated, skin parasthesias usually are elicited in the right lower quadrant and upper inner thigh, due to irritation of the ilioinguinal and iliohypogastric nerves. Often wth a low lying extensive abscess hypersensitiveness is present on the superior lateral aspect of the thigh, caused by irritation of the lateral cutaneous nerve. Actual swelling or edema of the lumbar region is a late sign.

In abscess about the upper pole of the kidney the diagnosis may be more difficult. Rigidity and tenderness are frequently absent, or present to a slight degree. If the abscess perforates the renal fascia and comes to underlie the diaphragm

the signs and symptoms of a subphrenic abscess are superimposed.

On admission all patients had fever, in 28 the daily rise was 101° or above. Twenty-five had a daily pulse rise to 100 or more.

TABLE VI

Daily elevations of temperature and pulse.

Degree of Temperature	Number of Patients	Pulse	Number of Patients
99-100	1	80-90	5
100-101	4	90-100	3
101-102	17	100-110	10
102-103	7	110-120	9
103-104	4	120-130	5
		130-140	1

Twenty-two of the 30 patients had a history of some degree of weight loss, nine losing over 20 pounds and 13 less than 20. The appetite was good in one, fair in 14 and poor in 18 cases.

TABLE VII

Degree of weight loss in 22 patients.

Pounds	Number of Patients
10-20	3
20-30	1
30-40	3
Marked weight loss	5
Some weight loss	9

The urinalysis is of little assistance in arriving at the diagnosis. Albumin was present in seven, pus cells in appreciable amounts in nine, and red cells in four.

A definite leucocytosis was present in the majority of the patients. Thirty had a white blood count ranging above 10,000 white cells. The lowest white blood count was 4,000 and the highest was 34,000. In 24 the differential polymorphonuclear count was above 80 per cent.

TABLE VIII

A study of the degree of leucocytosis.

White Blood Count	Number of Patients
4- 6,000	1
6- 8,000	1
8-10,000	1
10-12,000	2
12-14,000	6
14-16,000	6
16-18,000	3
18-20,000	5
20-22,000	5
28-30,000	2
32-34,000	1

Some degree of secondary anemia was present in 11 of the 30 patients.

TABLE IX

A study of the degree of anemia present.

Red Blood Count	Number of Patients
2-2,500,000	1
2,5-3,000,000	1
3-3,500,000	4
3,5-4,000,000	5
4-4,500,000	7
4,5-5,000,000	10
5-6,000,000	2

The blood chemistry was elevated in two cases, both having definite kidney pathology.

Urography, cystoscopy and ureteral catheterization are of more value in the differentiation of a kidney lesion. Retrograde pyelograms in 10 cases demonstrated on the side of the lesion: ureteral stricture in three, some distortion of calyces in two, upward displacement of kidney in one and negative findings in four. Intravenous pyelograms in eight cases showed on the involved side: poor function in four, upward displacement of the kidney in one, and negative findings in three. In the lateral view a forward displacement of the contrast filled kidney is helpful. Hilgenfeldt[2] emphasizes the value of the two films exposed at opposite extremes of the respiratory cycle for comparison of kidney mobility. Normal renal respiratory mobility varies from 1.5 to 6 cm. Mathe[3] stresses the importance of the lack of renal mobility as may be seen in the comparison of pyelograms taken in the supine and upright positions. Three centimeters is taken as a normal figure. An X-ray of the abdomen may show the kidney and psoas shadows to be indistinct or completely obliterated. Nine of 15 cases demonstrated this finding. Scoliosis of the lumbar spine with concavity toward the side of the lesion was less often present, being evident in seven of 15 X-rays. Occasionally the colon is displaced as is evident by the contained gas. Two such instances were recorded.

Dissimilar conditions may give rise to the erroneous impression of perinephric suppuration. Such pathology as colecystic disease, osteomyelitis of the twelfth rib, tuberculosis of the spine and hip, saccular aneurysm of the abdominal aorta, severe kidney infection, stone or malignancy, lumbar hernia, lumbar arthritis and retrocecal appendicitis may resemble a perinephric abscess in many respects.

No complications were encountered in

this series arising before incision and drainage. The danger of untoward extension of the neglected perinephritic abscess is ever present and is an added incentive for early diagnosis.

The prognosis of the operated perinephric abscess is good. The presence of kidney pathology will increase the operative hazard. One of the 30 operative patients expired. Simple incision and drainage was the procedure in all instances. In 19 the temperature was normal by the fifth postoperative day. No postoperative complications were encountered.

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New Books

PRINCIPLES OF HUMAN ANATOMY, With Synopsis and Bibliography. By Chas. F. DeGaris, M.D., Ph.D. (Hopkins), Professor of Anatomy in the University of Oklahoma School of Medicine, Oklahoma City, Oklahoma; Formerly Associate in Anatomy at Johns Hopkins Medical School, Baltimore, Md. Ernst Lachmann, M.D., (University of Breslau), Assistant Professor of Anatomy in the University of Oklahoma School of Medicine, Oklahoma City, Oklahoma. And Ralph E. Chase, A.M., (University of Oklahoma), Instructor in Anatomy in the University of Oklahoma School of Medicine, Oklahoma City, Oklahoma. 239 pages, Cloth, $2.50 net. Lea & Febiger, Philadelphia, 1939.

This compend on Human Anatomy, the authors of which belong to the anatomical department of the School of Medicine of the University of Oklahoma is prepared essentially for teaching purposes and of course will be of great value to the medical student, especially when preparing for examinations. It also makes an excellent reference book for the busy surgeon who wishes to look up some particular point. This of course sets forth the principles of anatomy as revealed by dissection of the human body and follows the course of anatomy as given by our Medical School.

CANCER OF THE BREAST and CANCER OF THE UTERUS by Marion Ellsworth Anderson, A.B., M.D., Clinton, Iowa. Second Edition.

This brochure of 100 pages on Cancer of the Breast and Cancer of the Uterus has been compiled from observations of the author during the past 30 years. It is not particularly adapted for the surgeon, gynecologist or pathologist; but to the general practitioner, a discussion of this subject in this form will benefit him greatly by its perusal. The bibliography is complete and many authorities are quoted.

The depth of the burn in the tissues is relatively not as important as the extent of skin surface involved.—Hygeia.

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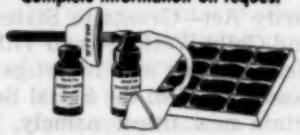

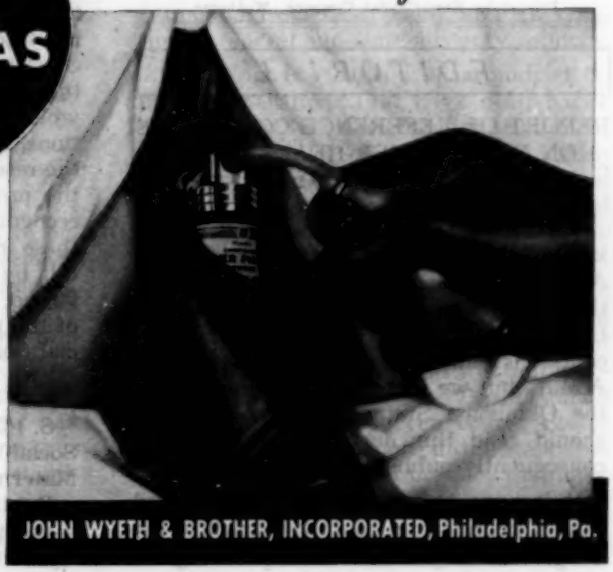

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McAlester, Oklahoma

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McAlester, Oklahoma

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Local news of possible interest to the medical profession, notes on removals, changes of addresses, births, deaths and weddings will be gratefully received.

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EDITORIAL

REPORT OF REFERENCE COMMITTEE ON THE WAGNER HEALTH BILL

By R. G. LELAND

There was considerable constructive legislation by the House of Delegates of the American Medical Association; however, the one outstanding action was taken relative to the Wagner Health Bill. This, of course, is published in the Journal of the A.M.A., but we feel that every member of the Oklahoma State Medical Association should read this document and we are consequently publishing it.

It is important that you read this and then it is important to do your part to see that the findings of the House of Delegates are carried out and each member can assist by bringing these findings to the attention of his Representatives and Senators. This is really constructive criticism.

* * * *

Your Reference Committee has carefully considered the Bill designated as S.-1620, "A Bill to provide for the general welfare by enabling the several states to make more adequate provision for public health, prevention and control of disease, maternal and child health services, construction and maintenance of needed hospitals and health centers, care of the sick, disability insurance, and training of personnel; to amend the Social Security Act; and for other purposes."

This bill was introduced by Senator Robert A. Wagner of New York, February 28, 1939, and is commonly referred to as the Wagner Health Bill. The bill itself provides that, if it be enacted, it may be cited as the "*National Health Act of* 1939." The purposes of the bill are sufficiently stated in the title, but the bill itself must be recognized as a proposed amendment to the Social Security Act of 1935. The bill is intended to make effective a national health program recommended by the Interdepartmental Committee to coordinate health and welfare activities.

The House of Delegates of the American Medical Association at its special session in Chicago, September 16, 1938, considered the National Health Program and adopted resolutions based on five recommendations contained in the program. It is important that this fact be borne in mind, for the bill, which was drafted long after these resolutions were adopted and at a time when the resolutions were presumably known to the proponents of this measure, does not recognize either the spirit or the text of these resolutions. Any criticism of this bill by the Association is not to be construed, therefore, as a repudiation of any of the principles adopted by the 1938 Special Session of the House of Delegates.

ANALYSIS OF THE BILL

S. 1620 proposes to amend Title V. of the Social Security Act—Grants to States for Maternal and Child Welfare—and Title VI —Public Health Work and Investigations —and proposes to add to the Social Security Act certain new titles: namely, Title

XII—Grants to States for Hospital and Health Centers; Title XIII — Grants to States for Medical Care, and Title XIV—Grants to States for Temporary Disability Compensation.

Already some individuals and organized groups in the United States have appeared before the Senate Subcommittee which has this bill under consideration and have urged its immediate enactment. Although the stated objectives of the Wagner Health Bill are generally recognized as desirable, your committee cannot approve the methods by which these objectives are to be attained.

Repeatedly, physicians and all other qualified professional groups have recommended the coordination and consolidation of the health activities of the Federal Government. The Wagner Health Bill leaves existing and proposed preventive and curative medical services widely scattered through several federal agencies.

This bill does not in any way safeguard the continued existence of the private practitioners who have always brought to the people the benefits of scientific research and treatment.

It does not provide for the use of the thousands of vacant beds now available in hundreds of church and community hospitals.

The Wagner Health Bill proposes an extensive program in the field of "health, diagnostic, and treatment centers, institutions and related facilities," without defining their functions.

This bill proposes to make federal aid for medical care the rule rather than the exception, since it does not specifically limit its benefits to persons unable to pay for adequate medical care.

The Wagner Health Bill does not recognize the need for suitable food, sanitary housing and the improvement of other environmental conditions necessary to the continuous prevention of disease and promotion of health.

This bill insidiously promotes the development of a complete system of tax supported governmental medical care, thus undermining and debasing present standards of medical services.

The House of Delegates in September, 1938, urged compensation for the loss of wages during sickness. The Wagner Health Bill deviates from this suggestion by proposing to provide medical services in addition to compensation.

The Wagner Health Bill would authorize an enormous expansion of governmental medical services and therewith ultimately unlimited appropriations for its health program. The funds necessary would be so great as to increase still further the present burdensome general taxation.

The Wagner Health Bill provides for supreme federal control. Rules and regulations must be promulgated by the Chief of the Children's Bureau in the Department of Labor, the Surgeon General of the Public Health Service, the Federal Emergency Administrator of Public Works, and the Social Security Board. These federal agents are given authority to disapprove plans proposed by the individual states.

The House of Delegates at its September, 1938, Session approved the expansion of preventive and other medical services when the need could be shown. The Wagner Health Bill prescribes no method for determining the nature and extent of the needs for which it proposes allotments of funds.

The provisions in the Wagner Health Bill that have never been considered by the House of Delegates are: the authorization of appropriations for studies, investigations and demonstrations, and the creation of federal and state advisory councils.

The Wagner Health Bill, as judged by the considerations that have been here presented, is inconsistent with the fundamental principles of medical care established by years of scientific professional medical experience, and in the opinion of your committee it is, therefore, contrary to the best interests of the American people.

For years the health of the people of the United States, as measured by sickness and death rates, has been better than that of most foreign countries, and this improvement has been continuous. The fortunate health conditions in the United States cannot be disassociated from the standards and methods of medical practice that have prevailed under the present system of medical practice.

No other profession and no other organi-

zation has done more for the prevention of disease, the promotion of health and the care of the sick than have the medical profession and the American Medical Association. No other groups have shown more genuine sympathetic interest in human welfare.

The contribution of the individual members of the American Medical Association to medical care is universally regarded as monumental in total volume. The contribution of the American Medical Association, through a program of medical education and the activities of its numerous councils which safeguard medical service, give abundant proof of interest in the problems of the national health. It has given continued consideration to these problems, whereas others show concern with these proposals because of a present but, it is to be hoped, a temporary need for relief. These are the groups which request revolutionary legislative action as indispensable for the extension and further diffusion of health facilities.

In view of its record and in consideration of the responsibility which American social history and the nature of medical care have imposed on the medical profession, the American Medical Association would fail in its public trust if it neglected to express itself unmistakably and emphatically regarding any threat to the nation's health and well-being.

The American Medical Association must therefore, speaking with professional competence, oppose the Wagner Health Bill.

Nevertheless, recognizing the soundness of the principles stated in the resolutions adopted by the House of Delegates at its special Session in 1938, namely, the expansion of preventive medicine and public health where need can be shown, the extension of medical care for the indigent and the medically indigent where the need can be demonstrated, with local determination of needs and local control of measures to supply these needs, your committee would urge the development of a mechanism for meeting these needs within the philosophy of the American form of government and without damage to the quality of medical services.

This question, as it relates to the aid to be given by an individual state to its own counties, municipalities or other local political units, is not immediately before this Association. The answer is to be found in the individual state constitutions and state statutes. Counties, townships and municipalities are creatures of the individual states and can be molded and guided by the state for its own purposes. The individual state, itself, is not a creature of the Federal Government. The Federal Government is, as a matter of fact, a creature of the individual states.

The fundamental question is how and when a state should be given financial aid by the Federal Government out of the resources of the states as a whole, pooled in the Federal Treasury. Disasters, such as floods, dust storms, fire and epidemics, have long been recognized as justifying such Federal aid. No state or person has ever been heard to object to the use of funds out of the Federal Treasury for such purposes. No one has ever proposed, however, that because Federal aid is extended under such conditions to a state in distress, a corresponding aid must be extended to every other state, regardless of its need. Nor has anyone ever been heard to say that Federal aid to a state in distress, because of flood, dust storm, fire or epidemic, shall not be extended, unless and until the suffering state has produced from its own treasury a stated amount of money to aid in affording the relief. The development of such bizarre thinking may be traced to those who have originated within comparatively recent years the granting of Federal subsidies—sometimes referred to as "grants in aid"—to induce states to carry on intrastate activities suggested frequently in the first instance by officers and employes of the Federal Government. The use of Federal subsidies to accomplish such federally determined activities has invariably involved Federal control. Any state in actual need of financial aid from the Federal Government for the prevention of disease, the promotion of health and the care of the sick should be able to obtain aid in a medical emergency without stimulating every other state to seek and to accept similar aid and thus to have imposed on it the burden of Federal control.

The mechanism by which this end is to be accomplished, whether through a Fed-

eral Agency to which any state in need of Federal financial assistance can apply, or through a new agency created for this purpose or through responsible officers of existing Federal Agencies, must be developed by the Executive and the Congress who are charged with these duties. Such method would afford to every state an agency to which it might apply for Federal assistance to enable it to care for its own people without involving every other state in the Union or the entire government in the transaction, and without disturbing permanently the American concept of democratic government.

SUMMARY

1. The Wagner Health Bill does not recognize either the spirit or the text of the resolutions adopted by the House of Delegates of the American Medical Association in September, 1938.
2. The House of Delegates cannot approve the methods by which the objectives of the National Health Program are to be obtained.
3. The Wagner Health Bill does not safeguard in any way the continued existence of the private practitioners who have always brought to the people the benefits of scientific research and treatment.
4. The Wagner Health Bill does not provide for the use of the thousands of vacant beds now available in hundreds of church and community general hospitals.
5. This Bill proposes to make federal aid for medical care the rule rather than the exception.
6. The Wagner Health Bill does not recognize the need for suitable food, sanitary housing and the improvement of other environmental conditions necessary to the continuous prevention of disease.
7. The Wagner Health Bill insidiously promotes the development of a complete system of tax supported governmental medical care.
8. While the Wagner Health Bill provides compensation for loss of wages during illness, it also proposes to provide complete medical service in addition to such compensation.
9. The Wagner Health Bill provides for supreme federal control: federal agents are given authority to disapprove plans proposed by the individual states.
10. The Wagner Health Bill prescribes no method for determining the nature and extent of the needs for preventive and other medical services for which it proposes allotments of funds.
11. The Wagner Health Bill is inconsistent with the fundamental principles of medical care established by scientific medical experience and is therefore contrary to the best interests of the American people.
12. The fortunate health conditions which prevail in the United States cannot be disassociated from the prevailing standards and methods of medical practice.
13. No other profession and no other group have done more for the improvement of public health, the prevention of disease and the care of the sick than have the medical profession and the American Medical Association.
14. The American Medical Association would fail in its public trust if it neglected to express itself unmistakably and emphatically regarding any threat to the national health and well being. It must, therefore, speaking with professional competence, oppose the Wagner Health Bill.
15. The house of Delegates would urge the development of a mechanism for meeting the needs for expansion of preventive medical services, extension of medical care for the indigent and the medically indigent, with local determination of needs and local control of administration, within the philosophy of the American form of government and without damage to the quality of medical service.
16. The fundamental question is how and when a state should be given financial aid by the Federal government out of the resources of the states as a whole, pooled in the Federal Treasury.

17. The bizarre thinking which evolved the system of Federal subsidies—sometimes called "grants - in - aid"—is used to induce states to carry on activities suggested frequently in the first instance by officers and employes of the Federal government.
18. The use of Federal subsidies to accomplish such Federally determined activities has invariably involved Federal control.
19. Any state in actual need for the prevention of disease, the promotion of health and the care of the sick should be able to obtain such aid in a medical emergency without stimulating every other state to seek and to accept similar aid, and thus to have imposed on it the burden of Federal control.
20. The mechanism by which this end is to be accomplished, whether through a Federal agency to which any state in need of Federal financial assistance can apply, or through a new agency created for this purpose or through responsible officers of existing Federal agencies, must be developed by the Executive and the Congress, who are charged with these duties.
21. Such a method would afford to every state an agency to which it might apply for Federal assistance without involving every other state in the Union or the entire government in the transaction.
22. Such a method would not disturb permanently the American concept of democratic government.

Transcript of Some Remarks Made by Dr. A. S. Risser before the House of Delegates at the State Meeting at Oklahoma City, May 3, 1939.

Mr. Speaker: It is my understanding that some 300 former members of the State Medical Association have not renewed their membership for the current year. This means that in a time of crisis, when we have had to make special efforts to prevent vicious legislation, when our expenses have been especially heavy, our income has been cut at the very outset some $3,000. (Besides these 300 there are almost 500 other physicians qualified to become members of our State Association but have never affiliated.) The members of our Legislative Committee, our Post Graduate Committee, the Councilors, and many other members in our organization besides paying their dues have given unstintedly of their time, have sacrificed their own professional business to further the work of the organization while these 300 and the 500 others have refused both personal service and the service which the payment of their dues would furnish. This condition, men of the House of Delegates, seems manifestly unfair. You here are giving of your own time and are sacrificing "business" at home to take care of the business of the whole organization while these 300 probably remain at home to profit by your absence and by what you do here for organized medicine. Again I say, it is unfair. Personally I have never admired hitch-hikers nor their prototype, the men who rode the rods and the blind baggage on the railroad without paying for their transportation. If there ever was a time when organized medicine needs *all* qualified men *in the ranks* it is *now*, when in our own State a mass of vicious legislation is threatening and when the federal government is straining every nerve to foist State Medicine upon us. More than the dues of these laggards, their money, we need their *man power*, educated, coordinated, united. Each man in his own office and among his own clientele to explain to his patients the evils of letting down the bars to cults and commercialism, of lowering medical standards, the loss they will suffer if they accept the subterfuge of state medicine. At home, "at the grass roots," if you please, is where we must do our educating and win our votes for the good both of the people and of the profession. If we leave it to our legislators, human as they are and susceptible to influences which we can not offer, we have already lost the fight for high standards of medical service. Each of you must be a missionary henceforth to win these men and others into the organization, to help educate them, that they may help to educate the laity, that our work and high calling may not have been in vain.

Editorial Notes—Personal and General

DR. FOWLER BORDER, Mangum, attended clinics in New York and Rochester during May.

DR. GEORGE H. KIMBALL, Oklahoma City, attended the A.M.A. in St. Louis. He visited the Plastic Clinics at Barnes Hospital.

DR. E. H. SHULLER, McAlester, has recently been confined in Albert Pike Hospital and is now convalescing from an appendectomy.

DR. G. A. KILPATRICK, McAlester, has returned from New York where he attended the International College of Surgeons' Meeting.

DR. HOUSTON SPANGLER, who has been Superintendent of Valley View Hospital at Ada has accepted a position with the American College of Surgeons.

DR. W. K. HAYNIE, Durant, has returned from New Orleans, where he took post graduate work. DR. J. T. WHARTON, Durant, is taking post graduate work on diseases of the heart in New York. DR. JOHN A. HAYNIE, Durant, will spend the months of June and July taking post graduate work in diseases of children in the New York Post Graduate Medical School of Columbia University.

The Meeting of the American Medical Association was largely attended by the doctors from Oklahoma. We were officially well represented as the President, President-Elect, Secretary-Treasurer-Editor, Executive-Secretary, two Delegates, and two Alternates were present and all were quite attentive to the Sessions of the House of Delegates. We received much valuable information as to the workings of this body, the things which have been accomplished by the A. M. A., both in a constructive way and obstructive to some of the legislative proceedings which did not appear to be to the best interests and health of the people of this country or to the interests of the medical profession.

Southern Oklahoma Medical Association held their Forty-third Quarterly Session Tuesday, June 6, 1939, at Sulphur, Oklahoma, State Veterans Hospital, under the auspices of Murray County Medical Society.

The following secientific program was heard: 1:30 p.m., registration—State Veterans Hospital. 1:45 p.m., "Cancer of the Uterus; Illustrated by Lantern Slides"—Dr. C. P. Bondurant, Oklahoma City; discussion by Dr. E. C. Lindley, Duncan. 2:30 p.m., "Goiter: Its Types and Treatment"—Dr. Arthur E. Hertzler, Halstead, Kansas; discussion by Dr. F. L. Carson, Shawnee. 3:30 p.m., "Colitis"—Dr. Tate Miller, Dallas, Texas; discussion by Dr. J. M. Gordon, Ardmore. 4:15 p.m., "Management of Arteriosclerotic Heart Disease"—Dr. Russel C. Pigford, Tulsa, Oklahoma; discussion by Dr. Wm. F. Dean, Ada.

Entertainment: 5:30 p.m., Artesian Hotel, Kiwanis Rooms. 6:30 p.m., Chicken Barbecue Dinner at the Vendome in Platt National Park, music by Don Percell's Colored Band. Entertainment Committee for visiting ladies: Mrs. W. D. DeLay, Mrs. Ernest Rose, Mrs. Arthur Fowler.

Oklahoma Physician Honored

Dr. Henry H. Turner, Oklahoma City, was elected president of the American Therapeutic Association which met in St. Louis, May 12th and 13th.

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OBITUARIES

DR. E. L. BAGBY

From "The Bulletin," Enid, Oklahoma

Dr. E. L. Bagby was born in Plattsburg, Missouri, March 13, 1875, and died in Enid, Oklahoma, May 10, 1939, after an extended illness from Mitral Stenosis and Myocarditis. Dr. Bagby was a graduate of the Ensworth Medical College, St. Joseph, Missouri, with the class of 1898. After graduation he located in Oklahoma, practicing his profession at Ralston and Fairfax, later accepting a position on the staff of the Eastern Oklahoma Hospital. On the entry of the United States in the World War he volunteered his services and was commissioned as Captain, Medical Corps. In recognition of his previous training and experience he was assigned to the Neuro-Psychiatric unit at Camp Gordon, which assignment he held until the close of the war. After his release from military service he returned to Oklahoma and was appointed superintendent of the Western Oklahoma Hospital and continued in that capacity until 1931. In that year, due to a change in the personnel of the state administration, he was again assigned to the staff of the Eastern Oklahoma Hospital and remained there until his appointment to the superintendency of the Northern Oklahoma Hospital at Enid in 1935.

The report of his stewardship of the Northern Oklahoma Hospital received special commendation from those in authority and attracted state-wide attention. It was shown to be the most efficient and economically managed of any state institution, and without the sacrifice on any part of the care, comforts or conveniences of the patients; on the contrary, all these were improved.

Early in 1939, realizing his health was failing, he entered a hospital in Kansas City, Missouri, for treatment. His condition gradually grew worse and realizing his days of usefulness were over he submitted his resignation to the Governor, put his worldly affairs in order and calmly awaited the event he knew was soon to come.

Dr. Bagby was a member of the Presbyterian Church, the Oklahoma State Medical Association, the Garfield County Medical Society, the Masonic Lodge at Fairfax, the Consistory at McAlester and the American Legion at Enid.

He is survived by his wife of the home, one daughter, Mrs. Milton Garber of Enid, and a brother, Dr. A. H. Bagby of Norman.

Funeral services were conducted at the First Presbyterian Church by the Rev. T. H. McDowell. Burial was in the Memorial Park cemetery, Enid.

Sleep—sleep on, Doctor!
Through the dark night, sleep on,
Until the Master bids you waken
To behold the glorious dawn.

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ABSTRACTS : REVIEWS : COMMENTS *and* CORRESPONDENCE

SURGERY AND GYNECOLOGY

Abstracts, Reviews and Comments from
LeRoy Long Clinic
714 Medical Arts Building, Oklahoma City

Experiences With Employment of Suction in the Treatment of Acute Intestinal Obstruction, A Reiteration of the Indications, Contra-Indications, and Limitations of the Method: O. H. Wangensteen, M.D., F.A.C.S., C. E. Rea, M.D., B. A. Smith, Jr., M.D., and H. C. Schwyzer, M.D.; Surgery, Gynecology and Obstetrics, May, 1939, Vol. 68, No. 5, page 851.

I was at the Meeting of the Western Surgical Association in Omaha in December, 1938, when this paper was read to the Association by Dr. Wangensteen.

Seven years ago Wangensteen reported the successful decompression of three instances of acute mechanical intestinal obstruction by continuous suction applied to an indwelling tube. He then indicated that the chief role of conservative decompression in the management of mechanical obstruction of the bowel would probably be found to be in instances of adhesive obstruction in which drainage of the bowel would permit automatic re-establishment of luminal continuity. He asserted at that time that colonic obstruction accompanied by great distention and strangulating obstructions were absolute contra-indications to attempts at achieving release of the obstruction by suction.

He explained in the initial communication seven years ago that the idea that the small bowel when obstructed could be decompressed by an indwelling duodenal tube was no accidental observation but was the outgrowth of quantitative determinations of the gas and fluid escape through enterostomy tubes following operative relief of obstruction. It was observed that as soon as drainage of the bowel proximal to the obstruction permits re-establishment of intestinal continuity, the escape of gas and fluid through the enterostomy usually ceases.

He now feels that the indications and the contra-indications for the employment of suction applied to an indwelling duodenal tube in the management of acute obstruction have remained essentially the same during these past seven years. At this time he feels it necessary to reiterate a warning in relation to the indications for the employment of suction. "The practice of employing suction as a test procedure to indicate whether operation will be necessary leads only to deferment of appropriate treatment." He feels that neglect of this admonition is the most mischievous error into which one may fall in the application of suction to patients with intestinal obstruction.

He freely admits that suction in such instances is a "blind method" far more dim sighted than "blind enterostomy." It is highly important, therefore, that he who uses the method be acquainted not only with its shortcomings, but also with the intricacies and limitations of diagnosis of acute abdominal disorders.

In this paper he sets forth the experience of his clinic with all types of mechanical obstruction of the small bowel over the seven-year interval between June 1, 1931, and June 1, 1938. The indications and contra-indications for the suction management of mechanical obstruction described in 1931 are reiterated and the lessons learned by him and his co-workers in the intervening years are reviewed. In this series among 156 patients treated for acute mechanical obstruction of the small intestine ,there were because of repetition of obstruction in some patients, 190 cases. There were 28 deaths in the entire group, a patient mortality of 17.9 per cent and a case mortality of 14.7 per cent.

In the group of cases in which suction was the primary treatment (though in a portion of these operation became necessary to effect a satisfactory decompression), there were 96 patients and 126 cases of obstruction. There were 15 deaths, a patient mortality of 15.6 per cent and a case mortality of 11.9 per cent. In those instances in which suction alone accomplished a satisfactory decompression and relief of the acute obstruction, there were 64 patients and 83 cases. Five deaths occurred, giving a patient mortality of 7.8 per cent and a case mortality of 6 per cent.

The suction group constituted 61.5 per cent by patient and 66.3 per cent by case of all patients treated for acute mechanical obstruction of the small intestine during the period under study. The number in which suction alone was the only direct attack employed for the relief of acute obstruction was 41 per cent by patient and 43.6 per cent by case of the entire group.

The conclusions reached by Wangensteen and his co-workers are as follows:

"Suction applied to an inlying duodenal tube has a definite role in the treatment of acute mechanical obstruction of the intestine. In suitable cases, complete relief of obstruction may be obtained through this agent alone. In a far larger group, suction is to be employed solely as an ancillary procedure, subordinate in importance to operative decompression or direct attack on the obstructive mechanism.

"The intelligent use of suction in the relief of acute intestinal obstruction rests upon the following:

"1. Ability to differentiate simple and strangulating types of obstruction.

"2. Ability to distinguish between acute obstruction of the large and small bowel.

"3. Appreciation of the importance of scout X-ray films for indicating the location of the obstruction and whether it is complete or incomplete, and for determining whether a satisfactory decompression is being obtained.

"4. Appreciation of the shortcomings of suction applied to an inlying duodenal tube in the management of acute intestinal obstruction.

"The experience of this clinic with the use of suction in the management of obstruction suggests that when employed on suitable indications and with full realization of its weaknesses and defects, it is a worth while addition to the available therapeutic agents, and that its rational use should lead

to a definite, general lowering of the mortality of acute intestinal obstruction.

"The conservative decompression mode of management is a painstaking procedure which demands meticulous attention to detail. The limitations of the method demand also that patients accepted for treatment be passed upon by trained observers. Frequent periodic resurvey of the status of a patient being treated conservatively is mandatory. The consistently best results in the treatment of acute intestinal obstruction will be obtained in hospital practice when the management of all such cases is concentrated in the hands of a few individuals who are interested in and willing to devote time and energy which such cases should rightfully command."

LeRoy D. Long.

Primary Streptococcus and Pneumococcus Peritonitis in Children. A Study of 61 Cases with the Report of Two Interesting Recoveries: Edward T. Newell, Jr., M.D., Chattanooga, Tennessee. Surgery, Gynecology and Obstetrics, April, 1939, Vol. 68, No. 4, page 760.

The article begins with the following statement: "The idiopathic or so called 'primary' cases of peritonitis, caused by the Beta streptococcus and the various types of the pneumococcus, while not common in children, occur with greater frequency than is generally recognized." The lack of uniformity of management is indicated, some believing in surgical operation, while others depend upon non-surgical procedures. A significant statement is that "the question of the portal of entry remains obscure although the point has been much discussed."

The study is based upon an analysis of the cases of patients in Johns Hopkins Hospital (Harriet Lane Home for Children), and from the department of surgery over a period of 25 years. During that time there were 25 patients who had primary streptococcus peritonitis and 36 primary pneumococcus peritonitis, these being proved by both bacteriological and pathological study. The mortality in the streptococcus group reached the astounding figure of 92 per cent, and in the case of the pneumococcus group the mortality was 61.1 per cent. There were autopsy studies in the case of 36 of the unfortunate patients who succumbed.

The author indicates that he prefers to abandon the classification of pneumococcus peritonitis into two groups, as has usually been done—that is, fulminating and subacute. He suggests that pneumococcus peritonitis be divided into three groups. The first one he would call "idiopathic," apparently because there is no demonstrable cause. In the group studied there were just 12 children. The mortality was 50 per cent. In the second group he includes patients where the peritonitis is associated with an upper respiratory infection. In the entire group upon which this analysis is based, there were eight patients in this class. In this connection, he says: "The upper respiratory infection usually preceded the peritonitis, but when pneumonia developed, the pneumonia was usually secondary to a fulminating pneumococcal peritonitis." In this group—that is, the second group in the classification employed by the author—the mortality was 62.5 per cent. The third group consisted of 16 patients who had nephrosis as a predisposing cause. The mortality in this group was 68.7 per cent.

The grouping suggested by the author seems to be a rational one.

In children, pneumococcus peritonitis occurs most often from the first to the tenth year, while streptococcus peritonitis occurs frequently from six months to two years.

The author has not observed that there is any particular predilection for the development of pneumococcus peritonitis in females.

Abdominal pain is the most frequent early symptom. It may or may not be diffuse. Quite often it may be noticed first in the right lower abdomen. "Other important symptoms are loss of appetite with nausea, and persistent vomiting; fever and diarrhea are also common."

As a rule patients are admitted to hospital from one to four days from the beginning of the attack. In this connection the author makes the following statement: "There is a small group of patients who have been seen two to six weeks following an attack of abdominal pain with vomiting and fever, and who, on examination, appear emaciated and have a large peritoneal abscess. These abscesses usually occur in the pelvic region, but sometimes occur in the epigastric region simulating a pancreatic cyst."

The most common finding is abdominal distention with tenderness on deep palpation. Muscular rigidity is not pronounced in the average patient. The temperature usually ranges from 101° to 104°, and the white blood count may be from 20,000 to 40,000.

In the doubtful case, it is advised that some material from the abdominal cavity be secured through the employment of a spinal puncture needle. The author says that the procedure has been described by Neuhof and Cohen, Danzer, Pollock, and others. He says that abdominal puncture as described by Neuhof and Cohen in 1926 is "simple and safe procedure." He emphasizes that it is necessary to secure only a drop or two of pus in order to make a differential diagnosis.

In connection with the management of pneumococcus peritonitis, it is advised that, in addition to surgical exploration, pneumococcus rabbit serum be administered. The cases of two children are reported, in one of which there was a total dose of 295,000 units of anti-pneumococcus rabbit serum, intravenously, at two-hour intervals over a period of 12 hours. This was the total dose, it being divided into fractions given intravenously every two hours. In the case of the other patient, 300,000 units of anti-pneumococcus rabbit serum was administered in fractional doses over a period of some hours. The last patient mentioned in this analysis was discharged from the hospital 24 days after admission, but the other patient had several severe complications that protracted the stay in hospital.

In connection with surgical treatment, it is indicated that it is not wise to undertake it until there has been localization.

To sum up, it appears to be the opinion of the author that when there has been a diagnosis of pneumococcus peritonitis operative procedure be deferred until there are distinct evidences of localization. The second interesting, and probably very important, observation made by the author is the remarkably satisfactory results following the intravenous employment of pneumococcus rabbit serum, as indicated in the case reports referred to above.

Streptococcus peritonitis is even more dangerous than pneumococcus peritonitis. Streptococcus peritonitis has been divided into the so-called "idiopathic" group and the group associated with erysipelas. In the group of so-called idiopathic streptococcus peritonitis studied by the author, there was a mortality of 88.2 per cent. In the erysipelas group there was a mortality of 100 per cent.

The author says: "The most important symptoms in the cases of streptococcus peritonitis in the order of their frequency are: vomiting, diarrhea, and abdominal pain. Many of these children are too young to reveal pain except by fretfulness and crying, particularly when the abdomen is touched.

"The more important signs are an acutely ill,

often moribund, child with considerable abdominal distention. The abdomen is tender to palpation but not board-like. In several of the cases the umbilicus and surrounding area for about two centimeters were red and swollen. Unfortunately, this is a late sign, which usually is seen in moribund infants. The children may have evidence of a streptococcus infection elsewhere, such as a pharyngitis, a cellulitis of the skin, or an otitis media. Leopold and Kaufman quote Mordlund's series of primary streptococcus peritonitis in which 63 per cent of the cases have a preceding upper respiratory infection."

The temperature varies from 103° to 106°, and the white blood count ranges from 2,700 to 45,000, the low count being seen most often in the moribund child.

It appears that conservative treatment of streptococcus peritonitis is the better procedure. It is suggested that sulfanilamide might be of considerable service.

LeRoy Long.

Statistical Analysis of One Thousand Abortions: Jalmar H. Simons, M.D., F.A.C.S.; American Journal of Obstetrics and Gynecology; May, 1939, Vol. 37, No. 5, page 840.

Though the accuracy of the data obtained from patients having abortion is recognized as unreliable, the author was able to draw the following conclusions:

"1. There is evidence of increasing number of abortions, principally in the self-induced group.

"2. Religion does not seem to be a deterrent to induction of abortion.

"3. As Taussig states, 'Abortion is a problem concerned with the married woman,' both induced and criminal, and it is in this group that the high death rate is obtained.

"4. Most spontaneous and induced abortions occur under three months and most often under 30 years of age, increasing again in incidence after 35.

"5. The total abortion incidence is one abortion to 2.7 pregnancies and one abortion to 1.6 confinements."

The mortality rate in this series was 1.9 per cent.

The conservative treatment was followed in the septic cases unless complicated by hemorrhage. In their septic patients evacuation of the uterus was done later if necessary when there was an approximately normal leucocyte count and temperature for a period of at least five days. They found the sedimentation time of little value in this respect.

Their nonseptic cases were evacuated, "almost routinely," in order to conserve blood and shorten hospitalization.

Repeated transfusions were of particular value in the infected patients and in those with hemorrhage. Practically all patients with a hemoglobin of 60 per cent or less received one or more transfusions. There is no statistical data given about sulfanilamide except that the author feels that it gives promise of great assistance in the septic patients.

The operative incidence in this series of 1,000 abortions was 51 per cent. This means that 51 per cent of the patients had an evacuation of the uterus.

In the discussion Dr. Ralph R. Wilson of Kansas City gave the statistical information of 1,200 abortion patients handled in the Kansas City General Hospital between the years 1923 and 1933 inclusive.

"1. The Kansas City series had an incidence of 25.3 per cent of septic cases as compared to 18.9 per cent in the Minenapolis cases.

"2. Death rate in the Kansas City series was 3.6 per cent as compared to 1.9 per cent for Minneapolis.

"3. Each city had one case due to Bacillus welchii and lesions in the uterus were obtained from an autopsy of the Kansas City case.

"4. Hemorrhage caused no deaths in the Minneapolis series while in the Kansas City series, four died from hemorrhage.

"5. Conservative, supportive, and symptomatic measures were the characteristic modes of treatment in both institutions."

There are two other interesting points in the discussion of this paper. The patients reported in the original article were examined by student clerks and interns and later by residents and the staff surgeon. Dr. J. L. Bubis questioned the advisability of so much manipulation.

The advisability of the use of pituitrin and other oxytocics was raised and it was Dr. Simons' opinion that pituitrin and ergot, especially pituitrin, might be harmful in inducting too much activity of the uterine muscle and thereby breaking down leucocytic barriers to infected areas.

Comments: As in all statistical reviews, there are certain sources of error, but, on the other hand, with a group as large as this one, it is possible to gain a splendid idea of the frequency of a condition and the merits of the various means and methods of treatment.

Taussig, who collected the available statistics on abortion in this country and abroad came to certain definite conclusions about proper treatment. It was evident from his statistics that the nonseptic cases did well with immediate evacuation. It was equally pronounced that the septic patients did not do well with immediate evacuation and that the complication and death rate was higher. The most important observation in this direction was to the effect that the septic and the nonsepic patients could not be differentiated when the patient first presented herself and treatment was instituted. He, therefore, came to the conclusion that expectant treatment was to be preferred until it was definitely demonstrated whether an individual patient could be classified in the category of septic or nonseptic. It is agreed by all that the septic patient should be treated conservatively and it is, I feel justly, concluded by Taussig that the nonseptic patients should be treated expectantly until they definitely demonstrate themselves to be nonseptic.

The use of ergot as an oxytocic has been frequently discussed and there have been theoretical objections, but from a practical standpoint it is my feeling that ergot is a very valuable drug in the treatment of abortion and should be employed. While there is the objection that it interferes with the leucocytic barrier and that it possibly pushes the infection in both directions, there is the equally potent theoretical argument that it contracts the uterus and forms a more solid barrier against invasion.

There would certainly be little argument about the advisability of infrequent manipulation of the pelvis. Pelvic examination should not be done for two or three days unless there is excessive hemorrhage after the patient has been placed in bed.

In this direction, many immediate evacuations are done upon the basis of hemorrhage on admission to the hospital whereas most of these patients will greatly reduce the amount of bleeding when placed in bed and kept quiet for a reasonable time.

Wendell Long.

Three Hundred Cases of Extensive Conization of the Cervix: Robert J. Crossen, M.D., and George J. L. Wulff, M.D.; American Journal of Obstetrics and Gynecology; May, 1939, Vol. 37, No. 5, page 849.

A modification of Hyams electrode was employed with the cutting wire on the electrode placed farther away from the central tube in order to permit the removal of all the infected tissue.

This series of 300 patients was operated upon by 12 members of the visiting staff of the Barnes Hospital and nine members of the house staff. In several instances the procedure was combined with radium therapy, supravaginal hysterectomy, and vaginal plastic operations.

In the 300 patients there were 17 who had postoperative bleeding and were all controlled satisfactorily by packing.

Both to control postoperative bleeding, and to encourage better healing, the authors feel that conization of the extensive type should preferably be done in the postmenstrual period so as to allow two or three weeks for healing before the onset of the following menstruation.

They also feel that the use of Sturmdorf suture anteriorly and posteriorly and if necessary laterally allows conization in the more extensive cases of chronic cervicitis where Sturmdorf operation would otherwise be the one of choice. They likewise feel that the suturing diminishes the possibility of postoperative bleeding and they condemn coagulation of bleeding points at operation as encouraging postoperative hemorrhage.

In their series of patients there was one stricture and there were three others with a tendency to stricture. The authors believe that a small rubber tube inserted into the cervical canal for seven days in the more extensive cases will prevent stricture.

In this group there have been 15 pregnancies following treatment with five full-term easy deliveries, seven miscarriages and three patients now pregnant.

Comments: This is an interesting contribution to cervical surgery and in conjunction with other reports such as that of Norman Miller emphasizes more strongly the fact that conization has a desirable place in the treatment of chronic cervicitis. Though it is not emphasized in this article, conization of the cervix in this extensive manner is entirely a hospital procedure. Conization cannot replace the other means of treating the cervix, but it has a peculiar adaptability in the treatment of certain selected patients.

Wendell Long.

EYE, EAR, NOSE AND THROAT

Edited by Marvin D. Henley, M.D.
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A Glareless Illuminated Holder for Visual-Acuity Test Charts with Variable Intensity of Light. C. E. Ferree, Ph.D., and G. Rand, Ph.D., Baltimore. American Journal of Ophthalmology, April, 1939.

This interesting article with its accompanying illustrations has the following summary by the authors.

Some of the faults in the present illuminated chart holders are: excessive glare from the lighting device; glare on the surface of the chart; a very uneven and poorly diffused illumination of the test surface; high light and brightness on the lateral edges of the chart and near to the illuminating units; an unstandardized and a too high intensity of light; and lack of portability.

The chart holder described in this paper was devised to remedy these defects. A simple mechanical provision is also made for varying the intensity of illumination in continuous change from approximately zero to full without change in the color, composition, or placement of the light.

The following are some of the situations in which there is an important need for such a chart holder. (a) In all cases where there is a problem of the accurate and reproducible testing of visual acuity; such, for example, as in motor-vehicle departments, in the testing of railroad employes, in the air service of the army and navy, and in the commercial air service. A particular and very important case of this need is in the testing of vision in the public schools, where customarily no attempt is made at a complete refraction correction and all knowledge of the pupil's eyes and all advances or recessions of any condition, refractive or otherwise, is dependent upon the testing of vision. Apparently this testing from the kindergarten through college is in a very unfavorable condition. A great variety of test equipment is being used and very little attention is paid to the standardization of the illumination of the test charts. Results obtained under these conditions are of little value from the standpoint of comparative ratings or the determination of anything approximating a set of norms. (b) In the correction of errors of refraction. By preference many refractionists still use and always will use a printed chart. The best test conditions, particularly for visual acuity, are given by a properly illuminated printed chart. With it, a better state of adaption may be had, a clearer definition of the test object, a better diffusion of light, and a better background for seeing the test object than can be had by any other type of test equipment. Provided with the feature of variable illumination, ideal conditions for testing vision and for detecting and correcting errors of refraction are obtained with the printed chart.

Role of the Tonsil in Experimental Endocarditis. Ira Frank, M.D., and Margery Blahd, M.D., Chicago. Archives of Otolaryngology, March, 1939.

"The advisability of removing the palatine tonsils as a prophylactic measure for the various forms of endocarditis has long been a subject of controversy among clinicians. The advocates of tonsillectomy point out the decreased incidence of chronic cardiac disease in tonsillectomized persons, as borne out by certain studies. Their opponents, basing their opinions on studies of other groups, maintain that routine removal of tonsils for the prevention of cardiac disease is not based on conclusive data. Furthermore, some clinicians contend that actual harm can be done by removal of the tonsils, not only in the operative mortality but also in respect to the development of infections of the respiratory tract."

Previously the results were reported by the author on 25 dogs which had intravenous injections of a virulent hemolytic streptococcus from a pneumonic lesion. Endocarditis developed in 40 per cent or 10 dogs. The method of transferring the infection is different in this group of 30 dogs.

In two of 30 dogs, after intratonsillar injection of virulent beta hemolytic streptococci acute endocarditis developed. The hemolytic streptococci used had previously produced endocarditis in 40 per cent of a series of 25 dogs. Endocarditis did not develop in any of 15 tonsillectomized dogs after repeated introduction of the same organism into the tonsillar bed and parapharyngeal regions. Furthermore, repeated blood cultures of the control dogs remained sterile. It is likely that the tonsils played a role in the development of bacteremia and endocarditis in these experiments. The results of the

investigations cannot be applied too readily to the patient. However, these studies present experimental confirmation of the favorable clinical results reported in the literature in respect to prophylactic tonsillectomy for endocarditis.

Electrocoagulation of Tonsils. Meyer L. Niedelman, M.D., Philadelphia. Annals of Otology, Rhinology and Laryngology, March, 1939.

This is a subject over which there has been and still is much controversy. The strongest advocates are those who have used this method and the bitterest critics are those who have not used the method at all or used it in an ill advised manner. The indications as listed by the author are:

1. General medical conditions such as hypertension, endocarditis, nephritis, tuberculosis, or any other chronic pulmonary conditions and syphilis. In this class can be included any condition which would contraindicate active surgery.

2. Neurotics who are in need of tonsillectomy but will not submit to surgery. This embraces a large class who, because of fear of hospitalization, hemorrhage or other complications, absolutely refuse to submit to surgery.

3. Blood dyscrasias, such as hemophilia, moderate secondary anemia, and long bleeding and coagulation time.

4. Economy to the patient. There are many patients, especially during the recent period of economic stress and strain, who cannot undergo hospitalization and the outlay of an immediate sum.

5. Postoperative tonsillar tabs or remnants and small lymphoid growths.

6. Those above the age of surgical risk.

7. Lingual tonsil and varix.

8. Carcinoma of the tonsil.

9. Public speakers and singers. There are no voice changes, as the vocal structures are left intact.

Contraindications

1. Children. The ideal here is obtained by general anesthesia, plus any of the recognized methods of enucleation.

2. For those who would rather have the work done at one sitting by surgical methods.

3. Acute or recent infections of throat or tonsils. Outside of these I know of no contraindications for electrocoagulation of the tonsils.

The author's conclusions are:

This is a safe and efficient method.

It does not and should not replace surgical removal, but is an alternative method in the removal of tonsils.

It is used when surgical removal is contraindicated.

A thorough knowledge of anatomy and surgical principles is prerequisite to the correct use of electrosurgical technique.

A method of anesthesia is outlined.

Two Cases of Brain Abscess Secondary to Acute Otitis Media, with Unusual Symptoms, Operation; Recovery. N. Asherson, London. The Journal of Laryngology and Otology, March, 1939.

Case one is a girl, age 5½ years. Left-sided otogenic temporo-sphenoidal lobe abscess. The following unusual features were noted.

1. The development of symptoms of intra-cranial pressure, following an attack of acute otitis media which had not given rise to any ear discharge—though the tympanic membrane was still inflamed, at the end of three weeks. At this time the symptoms were headache and vomiting, with papilloedema.

2. The slight changes in the cerebrospinal fluid, coupled with the papilloedema, suggested the diagnosis of a temporal lobe abscess. This was supported by the high polymorph white cell count.

3. The problem of the precise localization of the abscess was solved spontaneously by the brain and dura rapidly herniating though the first breach in the squama-exploration of this protrusion disclosed a large abscess.

4. The vagueness of any focal signs, in view of the large abscess disclosed, is a matter of comment. This suggests that the anterior part of the temporal lobe is a "silent" area in which a large abscess may collect insidiously. Hence this region must be exposed and explored when seeking for a suspected temporal lobe abscess in the absence of any definite localizing signs.

5. The abscess was exposed and drained through the squama, and not through the mastoid process.

Two and a half years later the child is still well.

Case two was in a child age three years. Acute temporal lobe abscess (right) secondary to an acute otitis media; with Jacksonian convulsions as the first and only symptom. Four days following a myringotomy a huge swelling developed suddenly and rapidly in front of the right ear. Comment on this case is as follows.

1. Until the onset of the convulsions and coma, no complication — inter-cranial or otherwise — was suspected. The brain abscess developed insidiously, and reached a large size without producing any symptoms to suggest its presence.

2. The location of the abscess. This was situated exactly beneath the superficial abscess which had developed on the fourth day after the myringotomy, in the temporal fossa. The dura was congested at this site and enabled the abscess to be easily located at the first incision.

3. The child was examined (after the temporal lobe abscess was drained) by Dr. C. P. Symonds, neurologist to the hospital. He looks upon the Jackson convulsions as due to an extension of the abscess to the cortex. He also notes that in such cases as this in which a Jacksonian convulsion is a leading symptom of the brain abscess, the convulsion is apt to recur at later dates, without the abscess necessarily pocketing or recurring.

4. When the second convulsion developed, it was unnecessary to reopen the wound as the temporal lobe could easily be explored by needle and syringe through the healed intact skin over it.

5. The brain abscess was drained through the squama.

Bilateral Endophthalmitis Complicating Pneumococcic Septicemia; Case Report. Jacob Reber, M.D., and Harold G. Scheie, M.D., Philadelphia. Archives of Ophthalmology, May, 1938.

The first manifestation of a generalized septicemia was the appearance of the eye infection. This is the report of a case of a man age 48, who on December 1, 1937, ran a tack in his foot (he was an upholsterer). The usual swelling and lymphangitis was treated, apparently successfully. Two weeks from the time of the accident he had a severe frontal headache and complained of a dimness of vision in each eye. Swelling followed the next day with more pain and a chill with an elevation of temperature. A consulting ophthalmologist found a typical endophthalmitis-corneas hazy-pupils small, irregular and fixed-posterior synechiae-iris hemorrhagic-fundus could not be seen—vision reduced to light perception. He was admitted to the hospital with a temperature of 103. The family and past history were of no importance. W.B.C. 20,500 with 82 polys. X-ray

showed some cloudiness in the ethmoids, which was ruled of no importance by the otolaryngologist. Other laboratory work was negative except the blood culture which showed type 16 pneumococcus. The patient ran a septic temperature (101°-105°). Culture from the nose showed staphylococcus aureus. The endophthalmitis gradually subsided —vision being only light perception. Medication consisted of sulfanilimide, daily transfusions and oral administration of ethylhydrocupreine hydrochloride, all of which were apparently of no avail. The patient died January 7, 1938. The blood culture remained positive. The autopsy showed among other things a phlebitis of the left femoral vein (the septic focus), the aspirated fluid obtained from the left cultured type 16 pneumococcus.

The unusual features of the case are that the ocular lesion was the first manifestation of a generalized septicemia and that there was an involvement of both eyes. According to Sherer systemic infections may show in the eye as iridocyclitis, metastatic ophthalmitis, septic retinitis of Roth and ring abscess of the cornea. In Groenouw's 166 cases reported in metastatic lesions of the eye from general sepsis, 85 per cent of the bilateral eye involvements were fatal.

"The physical factor for the production of the septic choroiditis or retinitis, as the case may be, is the lodging of the circulating organisms in either a ciliary or a retinal artery. These infected emboli set up a localized purulent inflammation, which, if it involves the coats of the eyeball and the surrounding orbital tissues, often results in spontaneous rupture of the sclera."

Some New Data Concerning the Pathology and Treatment of Ozaena. Dr. Geza Halasz, Budapest. The Journal of Laryngology and Otology, May, 1939.

This condition produces an inflammation and atrophy of the mucosa. Due to inadequate nutrition and degeneration the many glands in the nose become atrophic. There naturally follows that there is an insufficient supply of cleansing mucus in the nose. Then in the subsequent culture-media bacteria thrive and grow and there is present more pseudo-membranes and malodorous disintegration.

According to the author the ozaenous mucosa shows under the microscope a marked fibrosis, deficiency of cells, an additional amount of connective tissue and a shriveling of the turbinate bones. Mucous glands are diminished, the number of blood vessels are reduced, large collections of lymphocytes are to be found as well as hyaline and pigment-degeneration.

Atrophy is produced by general cachexia, inactivity, pressure and chemical injury but in this instance another factor enters, i.e. trophoneurotic troubles. The author demonstrated several years ago that in a pharyngitis after a tonsillectomy the infection was due mostly to an absence of a tonsillar endocrine function and could be cured by a subcutaneous administration of tonsillar extracts.

After a discussion of the relation of adenoids and tonsils in the increased amount of activity in a patient with ozaena, the author states that he believes that destruction or misdevelopment of the lymphatic ring is the primary cause of ozaena. He found that in his patients so afflicted the greater number had suffered from tonsillitis and subsequent pharyngitis in childhood and that their tonsils were scarcely visible, atrophic, pale and containing little lymphoid tissue. Adenoid vegetation was entirely absent. He states: "Hypoplasia of the tonsils and the adenoid tissue diminishes hormonal output, and the lack of hormone becomes manifest as ozaena."

After many experiments he prepared the following solution: Phyostigmini gta. 0.005, Potassi Sulf. 0.075, Acidi phosphorici 0.01, Aqu. dest. bister, ad 10.0, an isotonic solution. Injection of 0.5 c. cm. every day or every other day was made in each tonsil of the ozaena patient. The technique is described and is very simple as the injection is made directly into the tonsil. The result was: the nasal mucosa recovered its normal color and the vessels filled with blood; false membranes and foetor disappeared. The injections were made at longer intervals, a week or in some cases a month apart, after the patient was well under control. He likens the treatment to that of insulin in diabetics.

Many of the conflicting theories of ozaena are enumerated with their authors. The author's short summary is as follows:

1. Ozaena is caused by lack of tonsillar hormone.

2. Tonsils are specific endocrine glands, which regulate the vessels of the upper respiratory tract. The hormone is a substance similar to acetylcholine.

3. Successful treatment of ozaena has been achieved by means of intra-tonsillar injections of potassium and phyostigmine which inhibit the action of cholin-esterase.

A bibliography is appended.

—o—

PLASTIC SURGERY

Edited by
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Painless Rendering Closure of Superficial Wounds. Michael Gosis, South Ozone Park, N. Y., American Journal of Surgery, May, 1939.

The author presents a method of closing superficial wounds and lacerations which he has found practical and fundamentally sound. He makes no claim to priority of discovery but relates in simple detail the method which he states is an outgrowth of the time honored procedure of closing small superficial wounds with butterfly strips of adhesive. He states that the latter is not very applicable when one desires perfect approximation, for the broad base needed for the adhesive to adhere to the skin prevents the use of many strips placed close together as would be required. Further closing a wound with a broad piece of tape causes irritation of the skin edges which does not

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lend itself to the formation of hairline or almost invisible scars.

He states that out of 30 cases which he has done in the past two years, 29 of them had practically invisible resultant scars. After a few months during which time the initial erythema subsided, the scars were as satisfactory as those produced in plastic surgery.

The technique is described as used when there is no co-existent laceration of muscle or deep fascia. The wound is wiped dry and edges approximated. The adhesive is to be flamed before applying and then, skin over which it is to be applied should be painted with compound tincture of benzoin. The adhesive will adhere to the skin for at least 10 days with no appreciable loosening or irritation. The one-half inch strips of adhesive are placed one-fourth inch from the edge of the wound and extend to about three-fourths inch beyond the end of the wound. Wider strips may be used in case of longer wounds and narrower strips in case of small areas as nose and fingers.

The adhesive is then sutured with one continuous suture beginning outside the end of the laceration and running across from one strip of tape to the other the full length of the tape. It is best to have no elevation of the adhesive near the wound for insertion of needle and thread. Simply slip the needle between the skin and the superimposed tape without disturbing the bond between the two.

In irregular lacerations where sharp angles or corners of tissue must be approximated one might apply preliminary strips at the angles and then the tape as above outlined is sutured into place. Where the angle is not too acute strips may be placed parallel to the general direction of the wound edges without applying preliminary tension strips. Such lacerations may be about the fingers or palm of hand. In these cases the adhesive strips were placed parallel to the wound edges but they were made to hug the skin and invaginate at the creases. The first sutures taken in such situations are interrupted in order to approximate the crease marks, then the continuous suture is run along the length of the wound. After the sutures are in place it is sometimes advisable to place additional adhesive at right angles to the suture strips.

The author states that cleansing the wound with soap and water after picking out all gross contaminants and blood clots with thumb forceps, is sufficient. Where debris is found he states that a rubber drain may be placed into the depths of the wound and brought out at one end. This is left in for 24 hours. If debridement is necessary this method of wound closure is applicable, provided too much tissue is not removed. When deep tension sutures are not required for coaptation of skin edges suture of parallel adhesive strips with a broad base will serve as well.

The author states that when deep fascia and muscle lacerations are present requiring separate sutures, the wound cavity is filled with five per cent novocaine solution and the fluid allowed to remain there for about three minutes. This will anesthetize the deeper tissues and then the closure may be made in the manner described. Removal of sutures from five to seven days is the simplest procedure. The adhesive is loosened at one end and is removed sutures and all at one sweep. There is none of the pain due to tugging at the usual type of skin sutures.

Conclusion: This method is advocated for use in suitable wounds. Relieving the patient from unnecessary pain is adequate compensation for the extra precautions taken. Wound healing in cases not grossly infected at the time of injury is promoted by lessening the amount of additional trauma inflicted on a wound by penetrating sutures. Should infection ensue there is no obstacle to self-drainage. Suture removal in the event of a clean or infected wound is rendered absolutely painless.

Comment: The author apparently has very good results with this method of wound closure. The illustrations are adequate for anyone interested in the procedure. Personally I close wounds with sutures. The pain that is associated with closure can be avoided by local anesthetic and the pain of removing sutures is not worth mentioning in my opinion if horse hair or waxed silk is employed. Most people seem to have more fear of the procedure of suture removal because of what they have heard other epople say. It is certainly exaggerated.

Immediate Full Thickness Grafts to Finger Tips. Jewett V. Reed, M.D., F.A.C.S., and A. K. Harcourt, M.D., Indianapolis, Indiana. S. G. and O. May, 1939.

The authors describe a minor operation to be used in certain types of injury to the finger end which averts necessity for amputation or its alternative, thin skinned, painful scar. This operation is applicable and is used considerably in workingmens' hand injuries where there is loss of soft tissues of the finger end usually without loss of bone. The tissue loss may involve the distal tip, skin, and soft tissues sometimes exposing the bone. It may include one side of the whole distal phalanx sometimes involving the nail. It may remove the whole palmar pad.

The types of injury requiring the suggested graft to the finger tip are as follows: 1. Slice wound tip, not exposing bone. 2. Loss of one side of distal segment and nail. 3. Slice wound of pad without exposing bone. 4. Loss of soft tissues side of digit without injuring the nail. 5. Avulsion of pad of finger exposing palmar aspect of distal phalanx. 6. Loss of soft tissue exposing tip of phalanx.

The author has shown diagrammatic photos of each type above named and follows it with a chronological chart analysis of results in 53 cases, all patients being white males with two exceptions. Some of the injuries named are as follows: Mashed in steel girders, cut with steel chip pad amputated, cut with power saw, caught in joiner, caught in meat slicer, caught in gears, caught in punch press, caught in cogs, caught in bottle crowner, pinched between cable and drum, caught in food grinder, pinched in roller, etc.

A very comprehensive outline of the procedure used to transfer full thickness grafts to injured finger tips, is related by the authors. The suggested donor site in most cases is usually the volar aspect of the proximal fourth of the same forearm. In females the lateral aspect of the thigh

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near the ilium is chosen in an effort to place the scar where it will be at all times covered. The pattern is outlined with a sharp scalpel. Near the margin of the graft care must be taken to separate the skin from subcutaneous tissue accurately. Graft is immediately transferred to the injured finger and set into place by means of very small cutting edge needles and "A" silk. Sutures should be interrupted as continuous sutures choke off blood supply.

The patient is advised to keep the hand dependent a part of the time after a dry gauze quare is placed over the graft and held in place with a moderately tight bandage. The dressings are not changed for five days. Sutures not removed from the perimeter of the graft for two weeks. Usually at the end of five days the graft is pink and dry and appears similar to the surrounding skin except that fine hairs transferred in the graft, may be seen.

The authors state instances and results in the practice of the Indianapolis Industrial Clinic and give an analysis table from an economic viewpoint. Under the Indiana Compensation Law specific permanent impairment allowances are made for the loss of each digit.

Conclusions: An immediate full thickness graft applied to a finger end suffering soft tissue loss, conserves length and averts painful scar. The technique is simple. The end results are satisfactory to the individual, to the surgeon and to the person or to the company liable for the injury.

ORTHOPAEDIC SURGERY

Edited by Earl D. McBride, M.D., F.A.C.S.
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The April, 1939, issue of the American Journal of Surgery is devoted to a symposium on surgery of the extremities. It is the opinion of those abstracting that the information contained in this particular volume is invaluable to those interested in surgery of the extremities. It is impossible to abstract for the State Journal enough of the articles to be of more than brief interest. However, two articles will be abstracted to show primarily the value of the entire volume. These particular articles were chosen because this type of fracture is so frequently mistreated.

"Fractures of the Carpal (Navicular) Scaphoid," Edward K. Cravener, and Donald G. McElroy, Schenectady, N. Y. Amer. Jr. of Surg. Inc. New Series, Vol. XLIV, No. 1, April, 1939.

The authors try to estimate the frequency of carpal scaphoid or navicular fractures. They probably constitute around eight to nine per cent of all hand fractures. However, other writers give higher and some give lower incidences. The scaphoid bone is particularly valuable in that it is the wedge or keystone of the wrist arch. In the absence of this the wrist is very insecure. The bone is described as consisting of a body, constricting waist and a tuberosity, named in order from the proximal to the distal ends. The waist gives insertion to the radiocarpal ligaments and also to the blood vessels. This is particularly important in discussing healing.

Fractures through the tuberosities will heal even if somewhat neglected. Waist or body fractures are different and are the basis for the many treatments for fractures of the navicular. Many theories exist as to the cause of this; primarily that of embarrassed blood supply, or interposition of the radial-carpal ligament. However, the primary factor in union is complete prolonged immobilization.

In making a diagnosis of fracture of the navicular, first the X-ray is important. This is best when taken with the wrist slightly dorsiflexed. In this position the navicular is exposed its whole length and its overlying shadows are eliminated. This is done most easily with the fist clenched so that the wrist is kept slightly dorsiflexed and is firm. Any specific symptoms of navicular fracture are of no great importance since the advent of X-ray since any painful wrist after trauma should be submitted to X-ray examination.

Eight different types of fractures of this bone are described.

The authors recommended treatment as follows: In the case of tuberosity fractures, hold the wrist for three weeks in dorsiflexion and radial deviation. Fractures through the low waist should be kept in fixation at least eight weeks and then check for X-ray evidence for union before permitting free use of the wrist. In complicated old fractures through the upper waist or body, these must be exposed surgically and autogenous bone graft passed through from the distal to the proximal fragment. This has been modified some by making several drill holes through the fragments across the fracture line and pass the graft through one of these. This is not simple surgery and should not be undertaken by one not familiar with the anatomy of the wrist.

The authors feel that surgery is conservative treatment in this type of fracture in that the usual result will be a definitely painful wrist which may later require removal of a portion of the bone. They cite the fact that X-ray should be taken accurately and carefully. Plaster fixation is used, or possibly splint fixation only in tuberosity fractures. In low waist fractures, possibly plaster or splint fixation. In all high waist and body fractures drilling and bone pegging are used. In the case of a degenerated fragment resection is used. When speed and the assurance of a good result are desired, drilling and bone graft are advised except in fractures of the tuberosity, or in cases of degenerative arthritis.

"The Diagnosis and Treatment of Fractures of the Pelvis and Their Complications," Grover C. Weil, Edwin M. Price and Harold W. Rusbridge. Pittsburgh, Pa. Amer. Jr. of Surg. Inc. New Series Vol. XLIV, No. 1, April, 1939.

This fracture is not a rare fracture in modern transportation methods and is estimated to include three per cent of all skeletal fractures. Any disturbed continuity of the pelvis and its normal anatomical relationship causes severe changes in body function and every effort should be made to secure perfect reduction. The high mortality from 10 to 30 per cent causes these fractures to be placed in the realms of major injury.

The anatomy of the pelvis is reviewed briefly and the functions, both of the viscera and the body as a whole; standing and sitting.

The three particular points of weakness of the pelvis are: (1) midway between the symphysis pubis and the acetabulum; (2) the symphysis pubis; and (3) a trifle external to both sacroiliac joints.

In the simpler forms of fracture involving one of the rami on either side, or a moderate separation of the symphysis, without loss of continuity of the pelvic ring, only mild physical complaints may be recorded. With the history of some form of trauma, one can find tenderness over some portion of the pelvic ring particularly in the perineal region, over the adductor muscles and the most typical sign is that of pain on compression of the iliac crests. X-ray is positive evidence. Occasionally intestinal ileus occurs in mild injury. In severer forms of injury the physical signs include those in the pelvis plus those from injury to the viscera.

The symptoms and physical findings in such cases include shock in varying stages, inability to demonstrate proper function of one or both lower limbs by voluntary elevation while reclining. This attempt is always painful about the groin, inguinal and pubic regions. The bladder may be found distened and the abdomen may show distension with muscular resistance. Lateral compression of the iliac crests or hip joints imparts a giving sensation, showing a lack of firmness and crepitus may be present. Pain may be produced by palpation along the pelvic ring, depending on the location of the fracture. There may be beginning paralytic ileus, especially in severe types.

Rectal examination may show a fragment to press upon the rectal wall or a sharp spicule of bone may rupture into the rectum. There is either anuria or definite rupture of the urethra. In case of rupture there always appears some evidence of blood at the urethral orifice. There may be extravasation of hemorrhage or urine into the soft tissues. Gas bacillus infection, developing after rupture of the rectal walls, appears early and the presence of bluish-brown, discolored form of cellulitis of the skin and subcutaneous tissues, spreading over the perineum, scrotum and abdominal wall, with emphysema, point definitely to its presence.

The authors have found that good first aid care followed by immediate treatment of the fractures offers the best solution for prevention of shock and after effects. The patients are transported carefully in splints; they are brought to the hospital. After careful preliminary examination, X-ray is made and if injury is not too severe the patient is put to bed, traction is applied to the limb and hammock adjusted as is necessary for the type of fracture. Intestinal ileus is anticipated and the patient is usually given cascara pills followed by enema, consisting of soap suds, glycerin and sodium bicarbonate, in cases where not contraindicated. Sedatives are given for rest at night. For severe types of ileus the usual surgical procedures are adopted such as turpentine stupes, surgical pitrutrin, physostigmine, pitressin and prostigmine. Often more heroic methods are necessary, with the use of paravertebral or intraspinal injections of novocaine. Continuous oxygen inhalation is helpful.

In reduction of the severe fractures, X-ray is necessary to determine the exact procedure and treatment is carried out according to this.

The most frequent and important complications are: shock, ileus, hemorrhage, rupture of the rectum, urogenital injuries and infections. It is important that these complications be kept in mind and treatment instituted immediately on the diagnosis of any of them.

All patients, except those with very minor fractures, are confined to bed for a period of eight to ten weeks, after which the patient is confined to wheel chair for two weeks, before allowing gradual body weight. Physical therapy, such as infra-red, massage or diathermy is instituted, after the second week.

* * *

It is the opinion of the abstractors that the volume of the American Journal of Surgery mentioned above, should be in the library of every doctor who does traumatic surgery or surgery of the extremities.

"Gas-Bacillus Infection as a Complication of Fractures," David M. Bosworth, New York City. Jr. Bone & Joint Surg., Vol XX, No. 4, October, 1938.

The author reviews the history of gas-bacillus infection, particularly that of the men who have discovered the various organisms and its effect during the wars. The bacteriology is then discussed, followed by the pathology. Diagnosis of this condition is made on the odor, which may or may not be present. "The most outstanding impression is that of a very sick patient, slightly disoriented, complaining of great pain if asked, but otherwise lying quietly with a flushed face, very rapid pulse and no great temperature." The patient is sick and toxic way beyond the bounds indicated by temperature alone. Percussion of the skin near the wound area usually gives a tympanites and crepitus. Discoloration appears gradually and later X-ray examination shows gas in the soft tissues. This is not positive, however, unless an increase of gas is seen on successive films. A smear from the wound will show anaerobic bacteria, which is absolutely diagnostic.

Treatment: The author feels first that prophylactic doses of tetanus and gas antiserum should be given in every case of compound fracture. In case of a positive infection, curative serum therapy is commenced immediately before even the bacteriological diagnosis is positive. The author uses 20 c.c. doses of refined polyvalent serum intravenously every two hours until the infection is under control. No intramuscular serum has been used. If the case is operative, drainage is usually sufficient through the wound. If only a compound wound is present it should be enlarged through the skin and fascial planes with due regard for vessels and nerves. Extensive debridement or removal of muscles should be avoided. Repeated massive infusions of glucose and saline are routinely used. Support of the part in metal splints with continuous moist dressings to encourage drainage is strongly advised. The author states that amputation is senseless and harmful and all reports to date will bear this out. X-ray therapy has been reported favorably and the author thinks that cannot do harm and may do good. However, this should not take the place of the antiserum.

The author cites three cases. One case received a total of 400,000 units of the antiserum before he was successfully healed.

Comment: It still seems reasonable to us in this part of the country to amputate in gas gangrene infections. However, I believe that as we study our cases we will likely depend more on our serum and less upon surgery. The final word in this has not yet been stated.

UROLOGY

Edited by D. W. Branham, M.D.
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Preoperative Prostatic Mortality. W. J. Engel, Cleveland Clinic, Cleveland, Ohio. Journal of Urology, April, 1939.

Dr. Engle has critically analyzed 54 cases of prostatic obstruction who died before surgery was performed in an attempt to determine the predisposing factors in his preoperative management that may possibly have favored such a result.

In his opinion 40 or 74 per cent were what might have been classified as unavoidable but 14 or 26 per cent could have been prevented by a different technique of preoperative care. He believes that the dangers attendant with the first catheterization and instrumentation of the patient and also the inlying urethral catheter condemned as a routine procedure for preoperative drainage is underestimated.

He summarizes his plan of preoperative management under four headings:

1. Patients who are in excellent condition as far as blood nitrogen without complete retention and a small amount of residual urine have surgery performed without preliminary catheterization.

2. Patients who have complete retention but are

in good condition otherwise are intermittently catheterized for a short while before surgery is performed.

3. Those patients whose condition requires preliminary catheter drainage have a suprapubic vesical puncture made which is followed by the insertion of a catheter.

4. Inlying catheters for those in whom a suprapubic puncture is contraindicated, such as, a badly infected bladder.

Since this plan of management has been introduced the author states that not only the mortality rate has been reduced but patients have a smoother convalescence following surgery.

A Task Of All the People. Ray Lyman Wilbur, M.D. Journal of Social Hygiene, March, 1939.

Excerpts from an address by Dr. Wilbur before the Committee of Fifteen in Chicago, February 13, 1939.

"We have now reached the stage where we know something of the costs of syphilis and gonorrhea. We have adequate remedies for their treatment. We know procedures that can protect many of the innocent. The problem is simply one of how to get started with the proper campaign and how to carry it on over the years in order that these diseases may be made at least rare, rather than common."

"When we realize that 50 per cent of venereal patients acquire these diseases innocently, accounting for the congenital cases and other accidental infections, and that the other 50 per cent is acquired through either public or clandestine prostitution, and sexual promiscuity, we see that we face a problem where we ought to make every effort for the protection of youth, at least."

"The greatest single disseminator of these diseases is the prostitute. The tavern with its present freedom gives her easy refuge and makes it easy for her to entice boys and young men. There have been all sorts of schemes to control the so-called "women of the street." None of them have been completely satisfactory. Experience has shown that the one thing that is most important of all is to make her difficult of access. In that way there is the most likelihood of giving protection to youth. We cannot stop the sophisticate but we can protect minors. No scheme of segregation or of medical examination of the prostitute has stood up as of value in the control of the spread of the gonococcus and the spirochete. A certificate of health given to a prostitute may be a death certificate with a delayed reaction for the young man involved."

"We have been slipping back steadily for over a decade, have become more careless in the control of prostitution, have done less to protect our children, have a wider spread of unsavory literature, and have more alcohol being consumed by our boys and girls."

"We cannot turn this job over to the government alone. But every good citizen, parent, teacher, church member, has a responsibility at least to youth in seeing that they obtain satisfactory education and sufficient information on these important and prevalent diseases."

Effect of Alcohol on Normal Kidney and Kidney of Bright's Disease. Maurice Bruger, M.D., S. Arthur Localio, M.D., and Nobel W. Guthrie, M.D., New York. The Journal of the American Medical Association, May 6, 1939.

Twenty-one patients were studied of whom five were free from renal diease, five had acute diffuse glomerular nephritis and five had arteriosclerotic nephritis. Alcohol was administered to these individuals in appropriate amounts under a standardized routine as far as rest and dietary intake was concerned. Various laboratory examinations of the urine were made as well as several different renal functional tests were performed to evaluate the effect of alcohol on the kidney.

Their conclusions were:

"Alcohol or whiskey in moderate doses rarely augments the proteinuria of patients with Bright's disease. Moderate diuresis may be induced even in the presence of marked impairment of renal function. Alcohol or whiskey has no deleterious effect on the kidneys of normal subjects, nor does it aggravate the renal lesion in patients with acute or chronic diffuse glomerular nephritis. In patients with arteriosclerotic nephritis (nephrosclerosis) the ingestion of alcohol or of whiskey is followed frequently by a transient increase in the number of red and white blood cells and casts excreted in the urine and by a temporary diminution in renal function."

Physiological Bladder Changes During Pregnancy and the Puerperium. S. Richard Muellner. Journal of Urology, May, 1939.

The effects of pregnancy on the upper urinary tract have been repeatedly demonstrated. The possibility that pregnancy might produce similar changes in tone and capacity of the bladder suggested this study. Fifty normal women in varying stages of pregnancy were subjected to cystometric studies.

Their conclusions were:

"The bladder undergoes progressive changes in tone and capacity during pregnancy and the puerperium.

Beginning with the third month of gestation there is a gradual increase of capacity and decrease in tone, reaching extreme levels in the last trimester. The tone improves before termination of pregnancy.

The bladder empties without residual during pregnancy.

Post partum, the bladder capacity is apt to be largest, and there is considerable decrease in tone as compared with the ninth month. Residuals are common and may be quite large during this period, and should be drained by catheter if infected.

The bladder returns to normal over a period of six to eight weeks.

The parallelism between the bladder changes and those of the upper urinary tract is pointed out and its significance is discussed."

Comment: A study such as this develops knowledge of a practical nature that one may apply in general practice. Many intractable infections of the female bladder date from infection contracted during and immediately following pregnancy. These infections develop because the obstetrician pays insufficient attention to the bladder as far as its complete emptying is concerned.

Only Fifteen Minutes Required In New Syphilis Test Method

A new test, requiring only 15 minutes, for the demonstration of the syphilitic organism is reported by Leon Friedman, M.D., Philadelphia, in "The Journal of the American Medical Association" for January 14.

The test is especially applicable in cases of gonorrhea in which syphilis is suspected and, in comparison with other tests, saves considerable time.

The principle of the test is centrifugation of the gonorrheal discharge, or any other body fluid capable of being collected in a capillary tube, at low speed. This gives a clear specimen of serum suitable for dark field examination.

The author isolated the syphilitic organism from the gonorrheal discharge in one patient 50 days before the blood test showed the organism and in two patients whose history was not suggestive of syphilis.

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COX, J. L. ..... Ardmore
DARGATZ, F. E. ..... Newkirk
HARDY, WALTER ..... Ardmore
HATHAWAY, W. G. ..... Lone Grove
HIGGINS, H. A. ..... Ardmore
JACKSON, T. J. ..... Ardmore
JOHNSON, G. E. ..... Ardmore
JOHNSON, WALTER ..... Ardmore
LOONEY, MacDONALD ..... Marietta
MOXLEY, J. N. ..... Ardmore

PARRISH, R. M. ..... Ardmore
POLLOCK, J. R. ..... Ardmore
SULLIVAN, R. C. ..... Ardmore
VEAZEY, L. C. ..... Armore
VEAZEY, J. H. ..... Ardmore
VON KELLER, F. P. ..... Ardmore
WOODS, L. B. ..... Ardmore

CHEROKEE

ALLISON, J. S. ..... Tahlequah
BAINES, SWARTZ ..... Tahlequah
BARNES, HARRY E. ..... Tahlequah
BROWN, W. L. ..... Hulbert
DYER, ISADORE ..... Tahlequah
McINTOSH, R. K. ..... Tahlequah
MASTERS, HERBERT A. ..... Tahlequah
MATTHEWS, G. F. ..... State Health Dept., Oklahoma City
MEDEARIS, P. H. ..... Tahlequah

CHOCTAW

BOYER, H. L. ..... Ft. Towson
GEE, ROBT. L. ..... Hugo
HALE, C. H. ..... Boswell
JOHNSON, E. A. ..... Hugo
SWITZER, FRED D. ..... Hugo
WATERS, FLOYD L. ..... Hugo
WOLFE, REED ..... C.C.C. Camp, Oklahoma City

CLEVELAND

ATKINS, W. H. ..... Norman
BERRY, CURTIS ..... Norman
BOBO, C. S. ..... Norman
BUFFINGTON, F. C. ..... Norman
COOLEY, B. H. ..... Norman
DORSEY, ELIZABETH ..... Norman
FOWLER, W. A. ..... Norman
GABLE, J. J. ..... Norman
GRIFFIN, D. W. ..... Norman
HADDOCK, Jr., J. L. ..... Norman
HOOD, J. O. ..... Norman
HOWELL, O. E. ..... Norman
MAYFIELD, W. T. ..... Norman
MERRITT, I. S. ..... Norman
NIELSON, GERTRUDE ..... Norman
PROSSER, M. P. ..... Norman
RAYBURN, C. R. ..... Norman
REICHERT, R. J. ..... Moore
SCHMIDT, ELEANORA ..... Norman
STEEN, CARL ..... Norman
STEPHENS, E. P. ..... Norman
THACKER, R. E. ..... Lexington
TURLEY, L. A. ..... Norman
WICKHAM, M. M. ..... Norman
WILEY, G. A. ..... Norman
WILLARD, D. G. ..... Norman

COMANCHE

ANGUS, DONALD ..... Lawton
ANTONY, JOS. T. ..... Lawton
BARBER, GEO. S. ..... Lawton
BROSHEARS, JACKSON ..... Lawton
DOWNING, G. G. ..... Lawton
DUNLAP, E. B. ..... Lawton
FOX, FRED ..... Lawton
FERGUSON, L. W. ..... Lawton
GOOCH, L. T. ..... Lawton
HAMMOND, F. W. ..... Lawton
HATHAWAY, E. P. ..... Lawton
JOYCE, CHAS. W. ..... Fletcher
KERR, GEO. E. ..... Chattanooga
KNEE, L. C. ..... Lawton
LUTNER, THOS. R. ..... Lawton
MARTIN, CHAS. M. ..... Elgin
MITCHELL, E. BRENT ..... Lawton
PARSONS, O. L. ..... Lawton
VAN MATRE, REBER M. ..... Lawton

COTTON

BAKER, G. W. ..... Walters
COTTON, W. W. ..... Walters
HALSTEAD, A. B. ..... Temple
JONES, M. A. ..... Walters
SCISM, MOLLIE F. ..... Walters

CRAIG

ADAMS, F. M. ..... Vinita
BAGBY, LOUIS ..... Vinita
BRADSHAW, J. O. ..... Welch
DARROUGH, J. B. ..... Vinita
GASTINEAU, F. T. ..... Vinita
HAYS, P. L. ..... Vinita
HERRON, A. W. ..... Vinita
LEHMER, ELIZABETH ..... Vinita
MARKS, W. R. ..... Vinita
McMILLAN, J. M. ..... Vinita
McPIKE, LLOYD H. ..... Vinita
MITCHELL, R. L. ..... Veterans Hospital, Muskogee
SANGER, PAUL G. ..... Vinita
STAPLES, J. H. L. ..... Bluejacket
STOUGH, D. B. ..... Vinita
WALKER, C. P. ..... Grove

CREEK

BISBEE, W. G. ..... Bristow
COPPEDGE, O. S. ..... Depew
COWART, O. H. ..... Bristow
CROSTON, GEO. C. ..... Sapulpa
CURRY, J. F. ..... Sapulpa
HAAS, H. R. ..... Sapulpa
HOLLIS, J. E. ..... Bristow
JONES, ELLIS ..... Sapulpa
KING, E. W. ..... Bristow
LAMPTON, J. B. ..... Sapulpa
LEWIS, P. K. ..... Sapulpa
LONGMIRE, W. P. ..... Sapulpa
McDONALD, C. R. ..... Mannford
MOTE, PAUL ..... Sapulpa
NEAL, WM. J. ..... Drumright
REESE, C. B. ..... Sapulpa
REYNOLDS, E. W. ..... Bristow
REYNOLDS, S. W. ..... Drumright
SCHRADER, CHAS. T. ..... Bristow
SCHWAB, B. C. ..... Sapulpa
SISLER, FRANK H. ..... Bristow
SMITH, WENDELL L. ..... Drumright
STARR, O. W. ..... Drumright
WILLIAMS, J. CLAY ..... Neosho, Mo.
ZAMPETTI, H. A. ..... Drumright

CUSTER

ALEXANDER, C. J. ..... Clinton
BOYD, T. A. ..... Weatherford
CUNNINGHAM, CURTIS B. ..... Custer City
CUSHMAN, HARRY R. ..... Clinton
DEPUTY, ROSS ..... Clinton
DOLER, C. ..... Clinton
FRIZZELL, J. T. ..... Clinton
GOSSOM, K. D. ..... Custer City
HINSHAW, J. R. ..... Butler
LAMB, ELLIS ..... Clinton
LEWIS, R. W. ..... Clinton
LINGENFELTER, PAUL ..... Clinton
LOYD, E. M. ..... Taloga
McBURNEY, C. H. ..... Clinton
PAULSON, A. W. ..... Clinton
PIERSON, DWIGHT ..... Clinton
ROGERS, McLAIN ..... Clinton
RUHL, N. E. ..... Weatherford
SPEED, Jr., H. K. ..... Clinton
STOLL, A. A. ..... Clinton
ST. PETER, M. A. ..... Custer City
TISDAL, WM. ..... Clinton
TUTTLE, A. D. ..... Portalis, N. Mex.
VIEREGG, FRANK R. ..... Clinton
WOOD, J. G. ..... Weatherford

GARFIELD

BAGBY, E. L. ..... Enid
BAKER, R. C. ..... Enid
BITTING, B. T. ..... Enid
BRADY, J. H. ..... Fairview
CHAMPLIN, PAUL B. ..... Enid
DUFFY, FRANCIS M. ..... Enid
GREGG, O. R. ..... Enid
FEILD, JULIAN ..... Enid
HARRIS, D. S. ..... Drummond
HAMBLE, V. R. ..... Enid
HINSON, BRUCE R. ..... Enid
HOPKINS, P. W. ..... Enid
HUDSON, F. A. ..... Enid
HUDSON, H. H. ..... Enid
HYER, J. V. ..... Garber
JACOBS, R. G. ..... Enid
MAYBERRY, S. N. ..... Enid
McCROSKIE, M. R. ..... Fairview
McEVOY, S. H. ..... Enid
MERCER, J. WENDALL ..... Enid
METSCHER, ALFRED JOHN ..... Enid
MILES, G. O. ..... Enid
NEILSON, W. P. ..... Enid
NEWELL, JR., WALDO B. ..... Enid
NEWELL, W. B. ..... Enid
REMPEL, PAUL H. ..... Enid
RHODES, W. H. ..... Enid
ROBERTS, C. J ..... Enid
ROBERTS, D. D. ..... Enid
ROSS, GEORGE ..... Enid
ROSS, HOPE ..... Enid
RUDE, EVELYN ..... Enid
SHANNON, H. R. ..... Enid
SHAVER, S. R. ..... Fairview
SHEETS, MARION E. ..... Enid
TALLEY, EVANS E. ..... Enid
VANDEVER, H. P. ..... Enid
WALKER, JOHN R. ..... Enid
WATSON, J. M. ..... Enid
WILKINS, A. E. ..... Covington
WILSON, GEO. S. ..... Enid
WOLF, E. J. ..... Waukomis

GARVIN

ALEXANDER, ROBT. M. ..... Paoli
CALLAWAY, JOHN R. ..... Pauls Valley
GREENING, W. P. ..... Pauls Valley
GROSS, T. F. ..... Lindsay
JOHNSON, G. L. ..... Pauls Valley
LINDSEY, RAY H. ..... Pauls Valley
MONROE, HUGH H. ..... Lindsay
PRATT, CHAS. M. ..... Lindsay
ROBBERSON, JR., MARVIN E. ..... Wynnewood
ROBBERSON, M. E. ..... Wynnewood
SHI, A. H. ..... Stratford
SHIRLEY, EDWARD ..... Pauls Valley
SULLIVAN, C. L. ..... Elmore City

GRADY

BAZE, ROY E. ..... Chickasha
BAZE, W. J. ..... Chickasha
BONNELL, W. L. ..... Chickasha
BOON, U. C. ..... Chickasha
BYNUM, TURNER ..... Chickasha
COOK, W. H. ..... Chickasha
DOWNEY D. S. ..... Chickasha
EMANUEL, ROY ..... Chickasha
LEEDS, A. B. ..... Chickasha
LITTLE, AARON ..... Minco
MARRS, S. O. ..... Chickasha
MASON, REBECCA H. ..... Chickasha
McCLURE, H. M. ..... Chickasha
MITCHELL, C. P. ..... Chickasha
PYLE, OSCAR S. ..... Chickasha
RENEGAR, J. P. ..... Tuttle
WOODS, L. E. ..... Chickasha

GRANT

HARDY, I. V. ..... Medford
LAWSON, E. E. ..... Medford
LIVELY, S. A. ..... Wakita

GREER

BORDER, G. F. ..... Mangum
BROWN, F. R. ..... Mangum
HOLLIS, J. B. ..... Mangum
LANSDEN, J. B. ..... Granite
LOWE, J. T. ..... Mangum
MEREDITH, J. S. ..... Duke
OLIVER, W. D. ..... Mangum
PEARSON, LEB. E. ..... Mangum
POER, E. M. ..... Mangum
RUDE, JOE C. ..... Collis P. Huntington Memorial Hosp., Boston, Mass.

HARMON

HOLLIS, L. E. ..... Hollis
HOPKINS, S. W. ..... Hollis
HUSBAND, W. G. ..... Hollis
JONES, J. E. ..... Hollis
LYNCH, R. H. ..... Hollis
RAY, W. T. ..... Gould
STREET, O. J. ..... Gould
YEARGAN, W. M. ..... Hollis

HASKELL

CARSON, WM. S. ..... Keota
HILL, A. T. ..... Stigler
RUMLEY, J. C. ..... Stigler
WILLIAMS, N. K. ..... McCurtain

HUGHES

BENTLEY, J. A. ..... Allen
BUTTS, A. M. ..... Holdenville
DAVENPORT, A. L. ..... Holdenville
MAYFIELD, IMOGENE BUTTS ..... Holdenville
FORD, R. B. ..... Holdenville
HICKS, C. A. ..... Wetumka
HOWELL, H. A. ..... Holdenville
KIES, B. B. ..... Wetumka
MORRIS, R. D. ..... Allen
MUNAL, JOHN ..... Holdenville
PRYOR, V. W. ..... Holdenville
TAYLOR, W. L. ..... Holdenville
WALLACE, C. S. ..... Holdenville

JACKSON

ABERNETHY, EDW. A. ..... Altus
ALLGOOD, JOHN M. ..... Altus
BERRY, THOS. W. ..... Hugo, Colorado
BIRD, JESSE ..... 1545 N. W. 44th, Oklahoma City
BROWN, R. P. ..... Altus
CROW, E. S. ..... Olustee
ENSEY, J. E. ..... Altus
FOX, RAYMOND H. ..... Altus
HIX, JOSEPH B. ..... Altus
MABRY, EARL W. ..... Altus
McCONNELL, L. H. ..... Altus
REID, JOHN R. ..... Altus
SPEARS, C. G. ..... Altus
STARKEY, WAYNE ..... Altus
STULTS, J. S. ..... Altus
TAYLOR, ROBT. Z. ..... Blair

JEFFERSON

ANDRESKOSKI, W. T. ..... Ryan
BROWNING, W. M. ..... Waurika
COLLINS, D. B. ..... Waurika
DERR, J. I. ..... Waurika
EDWARDS, F. M. ..... Ringling
HOLLINGSWORTH, J. I. ..... Waurika
MAUPIN, C. M. ..... Waurika
McCALIB, D. C. ..... Ringling
WADE, L. L. ..... Ryan

JOHNSTON

LOONEY, J. T.	Tishomingo

KAY

ARMSTRONG, W. O.	Ponca City
ARRENDELL, C. W.	Ponca City
BARKER, W. JACKSON	Ponca City
BEATTY, J. H.	Tonkawa
CLIFT, M. C.	Blackwell
CURRY, JOHN R.	Blackwell
GARDNER, C. C.	Ponca City
GHORMLEY, J. G.	Blackwell
GIBSON, R. B.	Ponca City
GORDON, D. M.	Ponca City
GOWEY, H. O.	Newkirk
HOWE, J. H.	Ponca City
HUDSON, J. O.	Braman
KENSINGER, R. R.	Blackwell
KREGER, G. S.	Tonkawa
MALL, W. W.	Ponca City
MATTHEWS, DEWEY	Tonkawa
McELROY, THOMAS	Ponca City
MILLER, D. W.	Blackwell
MOORE, G. C.	Ponca City
MORGAN, L. S.	Ponca City
NEAL, L. G.	Ponca City
NIEMANN, G. H.	Ponca City
NORTHCUTT, C. E.	Ponca City
NUCKOLS, A. S.	Ponca City
OBERMILLER, R. G.	Ponca City
PETERS, M. L.	Blackwell
RISSER, A. S.	Blackwell
SANGER, W. W.	Ponca City
VANCE, L. C.	Ponca City
WAGGONER, E. E.	Tonkawa
WAGNER, J. C.	Ponca City
WALKER, I. D.	Tonkawa
WHITE, M. S.	Blackwell
WRIGHT, L. I.	Blackwell
YANDELL, HAYS R.	Ponca City
YEARY, G. H.	Newkirk

KINGFISHER

DIXON, A.	Hennessey
HODGSON, C. M.	Kingfisher
LATTIMORE, F. C.	Kingfisher
TAYLOR, JOHN R.	Kingfisher

KIOWA

ADAMS, J. L.	Hobart
ADAMS, RICHARD	Hobart
ANDERSON, H. R.	Mountain View
BONHAM, J. M.	Hobart
BRAUN, J. P.	Hobart
FINCH, J. WM.	Hobart
FREEMAN, W. H.	Sentinel
HATHAWAY, A. H.	Mountain View
MOORE, J. H.	Hobart
RITTER, J. M.	Roosevelt
WALKER, F. E.	Lone Wolf
WATKINS, B. H.	Hobart

LE FLORE

BAKER, F. P.	Talihina
BEVILL, S. D.	Poteau
BOOTH, G. R.	LeFlore
BRADLEY, FRANK	Talihina
COLLINS, E. L.	Panama
DEAN, S. C.	Howe
FAIR, E. N.	Heavener
HARTSHORNE, G. E.	Spiro
LOWREY, R. W.	Poteau
MINOR, R. W.	Williams
ROGERS, G. A.	Talihina
ROLLE, NEESON	Poteau
SHIPPEY, W. L.	Poteau
WOODSON, O. M.	Poteau
WRIGHT, R. L.	Poteau

LINCOLN

ADAMS, J. W.	Chandler
BAILEY, C. H.	Stroud
BAUGH, HAROLD	Meeker
BROWN, BYRON B.	Davis
BROWN, F. C.	Sparks
BURLESON, NED	Prague
ERWIN, PARA	Wellston
HURLBUT, E. F.	Meeker
MARSHALL, A. M.	Chandler
NICKELL, U. E.	Davenport
NORWOOD, F. H.	Prague
ROBERTSON, C. W.	Chandler
ROLLINS, J. S.	Prague

LOGAN

*BARKER, C. B.	Guthrie
BARKER, PAULINE	Guthrie
CORNWELL, N. L.	Coyle
DRESBACH, H. V.	Mulhall
FIRST, F. R.	Crescent
GARDNER, P. B.	Guthrie
HAHN, L. A.	Guthrie
HILL, C. B.	Guthrie
LARKIN, W. H.	Minco
LE HEW, Jr., J. L.	Guthrie
MEAD, W. W.	Guthrie
MILLER, WM. C.	Guthrie
PETTY, C. S.	Guthrie
PETTY, JAMES	Guthrie
RINGROSE, R. F.	Guthrie
RITZHAUPT, L. H.	Guthrie
SOUTER, J. E.	Guthrie

*Deceased

MAJOR

SPECHT, ELSIE	Fairview

MARSHALL

HOLLAND, J. L.	Madill
RAFF, JOS. S.	Madill
YORK, J. F.	Madill

MAYES

ADAMS, SYLBA	Hayward, Wis.
HERRINGTON, V. D.	Pryor
MORROW, B. L.	Salina
PUCKETT, CARL	22 W. Sixth, Oklahoma City
WERLING, E. H.	Pryor
WHITAKER, W. J.	Pryor
WHITE, L. C.	Adair

McCLAIN

COCHRANE, J. E.	Byars
DAWSON, O. O.	Wayne
KOLB, I. N.	Blanchard
McCURDY, W. C.	Purcell
ROYSTER, R. L.	Purcell

McCURTAIN

BARKER, N. L.	Broken Bow
CLARKSON, A. W.	Valliant
LOKEY, J. P.	Idabel
McBRAYER, W. H.	Haworth
McCASKILL, W. B.	Idabel
MORELAND, J. T.	Idabel
MORELAND, W. A.	Idabel
OLIVER, R. B.	Idabel
SHERRILL, R. H.	Broken Bow
SIZEMORE, PAUL	Broken Bow
WILLIAMS, R. D.	Idabel

McINTOSH

JACOBS, L. I.	Hanna
LITTLE, D. E.	Eufaula
ROY, EMILE	Checotah

STONER, RAYMOND WARD	Checotah
TOLLESON, W. A.	Eufaula
WOOD, JAS. L.	Eufaula

MURRAY

ANADOWN, P. V.	Sulphur
BALL, ERNEST	Sulphur
BROWN, BYRON B.	Davis
BURKE, RICHARD M.	Sulphur
DeLAY, W. D.	Sulphur
FOWLER, Jr., A.	Sulphur
LUSTER, J. C.	Davis
PARKER, WARREN E.	Davis
POWELL, W. H.	Sulphur
ROSE, ERNEST	Sulphur
SADLER, F. E.	Sulphur
SLOVER, GEO.	Sulphur

MUSKOGEE

BALLANTINE, H. T.	Surety Bldg.
BERRY, W. D.	Barnes Bldg.
BRUTON, L. D.	Commercial Natl. Bldg.
COACHMAN, E. H.	Manhattan Bldg.
DIVINE, DUKE G.	Wagoner
DORWART, F. G.	Barnes Bldg.
EWING, F. W.	Surety Bldg.
FITE, E. H.	Barnes Bldg.
FITE, W. P.	Barnes Bldg.
FRYER, S. J.	Surety Bldg.
FULLENWIDER, C. M.	Barnes Bldg.
HAMM, S. G.	Haskell
HOLCOMB, R. N.	Surety Bldg.
JOBLIN, W. R.	Porter
KAISER, GEO. L.	562 No. Sixth
KLASS, O. C.	Surety Bldg.
KUPKA, J. F.	Haskell
McALISTER, L. S.	Barnes Bldg.
MOBLEY, A. L., Vets. Facility,	Albuquerque, N. M.
NEWHAUSER, MAYER	Vet. Administration
NEELY, S. D.	Commercial Natl. Bldg.
OGLESBEE, C. L.	County Health Unit
OLDHAM, Jr., I. B.	426 North Sixth
OLDHAM, Sr., I. B.	426 North Sixth
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SAUNDERS, H. V.	437 Post Office Bldg.
SCOTT, H. A.	Commercial Natl. Bldg.
THOMPSON, M. K.	Surety Bldg.
TILTON, W. B.	Vet. Hospital, Muskogee
WARTERFIELD, F. E.	Commercial Natl. Bldg.
WEAVER, W. N.	Barnes Bldg.
WHITE, CHAS. ED	2430 Boston
WHITE, J. H.	Surety Bldg.
WOLFE, I. C.	426 North Sixth
WOODBURN, JOEL T.	Surety Bldg.

NOBLE

COLDIRON, D. F.	Perry
COOKE, C. H.	Perry
EVANS, A. M.	Perry
FRANCIS, J. W.	Perry
HEISS, J. E.	Perry
RENFROW, T. F.	Billings
WIGNER, R. H.	Marland

NOWATA

KURTZ, R. L.	Nowata
LANG, S. A.	Nowata
PRENTISS, H. M.	Nowata
ROBERTS, S. P.	Nowata
SCOTT, M. B.	Delaware

OKFUSKEE

BLOSS, JR., C. M.	Okemah
BOMBARGER, C. C.	Paden
BRICE, M. O.	Okemah
COCHRAN, C. M.	Okemah
JENKINS, W. P.	Okemah
LUCAS, A. C.	Castle
MELTON, A. S.	Okemah
PEMBERTON, J. M.	Okemah
PRESTON, J. R.	Weleetka
SPICKARD, L. J.	Okemah
WRIGHT, H. L.	Okemah

OKLAHOMA

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ALFORD, J. M.	Medical Arts Bldg.
ALLEN, E. P.	1200 North Walker
ALLEN, GEO. T.	1200 North Walker
ANDREWS, LELIA E.	1200 North Walker
ARRINGTON, C. T.	715½ North Walnut
BAILEY, F. M.	CCC Camp, High Rolls, N. M.
BAILEY, WILLIAM H.	301 West Twelfth St.
BAIRD, Jr., W. D.	2519½ S. Robinson
BALYEAT, RAY M.	1200 North Walker
BARB, THOS. J.	240 West Commerce
BARKER, CHAS. E.	505-13 Osler Bldg.
BARRY, G. N.	801 N.E. 13th
BATCHELOR, J. J.	Medical Arts Bldg.
BATES, C. E.	Vet. Admin., Dearborn, Mich.
BAUM, E. ELDON	Medical Arts Bldg.
BELL, A. H.	300 West 12th St.
BERRY, CHAS. N.	Medical Arts Bldg.
BEYER, M. R.	2006 N.W. 39th
BINKLEY, J. G.	Medical Arts Bldg.
BINKLEY, J. SAM	Medical Arts Bldg.
BIRGE, JACK P.	Ramsey Tower
BOATRIGHT, LLOYD C.	Perrine Bldg.
BOGGS, NATHAN	Perrine Bldg.
BOLEND, REX	Medical Arts Bldg.
BONDURANT, C. P.	Medical Arts Bldg.
BONHAM, WM. L.	Medical Arts Bldg.
BORECKY, GEORGE L.	Ramsey Tower
BORDER, C. L.	American Nat'l. Bldg.
BOWEN, RALPH	1200 North Walker
BRADLEY, H. C.	Perrine Bldg.
BRANHAM, D. W.	Medical Arts Bldg.
BREWER, A. M.	Perrine Bldg.
BROWN, G. W.	Medical Arts Bldg.
BRUNDAGE, C. L.	1200 North Walker
BURTON, JOHN P.	Osler Bldg.
BUTLER, H. W.	1200 North Walker
CAILEY, LEO F.	Medical Arts Bldg.
CAMPBELL, COYNE H.	719 No. Robinson
CATES, ALBERT	Medical Arts Bldg.
CAVINESS, J. J.	Medical Arts Bldg.
CHARNEY, L. H.	Medical Arts Bldg.
CLARK, ANSON L.	Medical Arts Bldg.
CLOUDMAN, H. H.	Medical Arts Bldg.
CLYMER, CYRIL E.	Medical Arts Bldg.
COLEY, A. J.	Hightower Bldg.
COLEY, JOE. H.	Hightower Bldg.
COLLINS, H. D.	Medical Arts Bldg.
COLLOPY, PAUL J.	State Health Dept.
COLONNA, PAUL C.	800 E. 13th
COOPER, F. M.	Medical Arts Bldg.
COSTON, TULLOS O.	Medical Arts Bldg.
CUNNINGHAM, JOHN A.	209 N.W. 13th
DAILY, H. J.	Medical Arts Bldg.
DANIELS, HARRY A.	610 N.W. 9th St.
DEMAND, F. A.	1200 North Walker
DERSCH, WALTER H.	Medical Arts Bldg.
DICKSON, GREEN K.	1200 North Walker
DILL, FRANCIS E.	Medical Arts Bldg.
DOUDNA, HUBERT E.	800 N.E. 13th
DOUGHERTY, VIRGIL F.	Sudan, Africa
DOWDY, THOS. W.	Medical Arts Bldg.
EARLY, R. O.	Medical Arts Bldg.
EASTLAND, WM. E.	Medical Arts Bldg.
ELEY, N. P.	400 West Tenth St.
EMENHISER, Jr., LEE K.	Medical Arts Bldg.
ERWIN, F. B.	Medical Arts Bldg.
ESKRIDGE, J. B.	1200 North Walker
FAGAN, HERMAN	400 N.W. 10th
FARIS, BRUNEL D.	Medical Arts Bldg.

FELTS, GEO. R. ..... Osler Bldg.
FERGUSON, E. G. ..... Medical Arts Bldg.
FERGUSON, E. S. ..... Medical Arts Bldg.
FISHMAN, C. J. ..... 132 West Fourth St.
FITZ, R. G., Taming Fu Hoppi, Prov. No. China
FLESHER, THOS. H. ..... Edmond
FOERSTER, HERVEY A. ..... Medical Arts Bldg.
FORD, HARRY C. ..... Medical Arts Bldg.
FRIERSON, S. E. ..... Medical Arts Bldg.
FULTON, CLIFFORD C. ..... Medical Arts Bldg.
FULTON, GEO. S. ..... American Natl. Bldg.
GALLAGHER, C. A. ..... 610 N.W. 9th
GARRISON, GEO. H. ..... 1200 North Walker
GEE, O. J. ..... Medical Arts Bldg.
GIBBS, ALLEN G. ..... Ramsey Tower Bldg.
GLOMSET, JOHN L. ..... 621 Osler Bldg.
GOLDFAIN, E. ..... Medical Arts Bldg.
GOODWIN, R. Q. ..... Medical Arts Bldg.
GRAHAM, ALLISON T. ..... 26 S.W. 25th
GRAY, FLOYD ..... 1200 N. Walker
GRAY, J. WORTH ..... 1315 South Agnew
HALL, CLARK H. ..... Medical Arts Bldg.
HAMMONDS, O. O. ..... 623 North East 18th St.
HARBISON, FRANK ..... Terminal Bldg.
HARBISON, J. E. ..... Terminal Bldg.
HARRIS, HENRY W. ..... 1200 N. Walker
HASKETT, PAUL E. ..... Hales Bldg.
HASSLER, GRACE CLAUSE ..... Med. Arts Bldg.
HATCHETT, J. A. ..... Medical Arts Bldg.
HAYES, BASIL A. ..... 625 North West 10th St.
HAZEL, O. G. ..... 1200 N. Walker
HEATLEY, JOHN E. ..... Medical Arts Bldg.
HERRMANN, JESS ..... Medical Arts Bldg.
HETHERINGTON, A. J. ..... 2014 Gatewood Ave.
HICKS, F. B. ..... Medical Arts Bldg.
HIRSHFIELD, A. C. ..... Medical Arts Bldg.
HOLLIDAY, J. R. ..... 1200 N. Walker
HOOD, F. REDDING ..... 1200 North Walker
HOOT, M. P. ..... Bartlesville, Okla.
HOWARD, R M. ..... Osler Bldg.
HUGGINS, J. R. ..... Medical Arts Bldg.
HULL, WAYNE M. ..... 800 N.E. 13th
HUNTER, GEO. ..... State Health Dept.
HYROOP, GILBERT L. ..... Medical Arts Bldg.
ISHMAEL, WM. K. ..... 605 N.W. 10th
JACKSON, A. R. ..... 2528½ South Robinson
JACOBS, MINARD F. ..... Medical Arts Bldg.
JANCO, LEON ..... 10 West Park Place
JETER, HUGH ..... 1200 North Walker
JOLLY, W. J. ..... 615 West 14th St.
JONES, HUGH ..... Medical Arts Bldg.
KELLER, W. F. ..... Medical Arts Bldg.
KELSO, JOSEPH W. ..... Medical Arts Bldg.
KELTZ, BERT F. ..... Medical Arts Bldg.
KERNODLE, S. E. ..... First Natl. Bldg.
KIMBALL, G. H. ..... Medical Arts Bldg.
KUHN, JOHN F. ..... Medical Arts Bldg.
KUHN, Jr., JOHN F. ..... Medical Arts Bldg.
LACHMANN, ERNST ..... 801 East 13th St.
LAMB, JOHN H. ..... Medical Arts Bldg.
LAMBKE, PHIL M. ..... 605 North East 28th St.
LaMOTTE, GEORGE A. ..... Colcord Bldg.
LANGSTON, WANN ..... Medical Arts Bldg.
LAWSON, PAT ..... 818 N.W. 11th
LEMON, C. W. ..... Medical Arts Bldg.
LENNY, FANNIE LOU ..... 400 N.W. 10th
LEONARD, C. E. ..... Supply, Okla.
LINDSTROM, W. C. ..... Medical Arts Bldg.
LINGENFELTER, F. M. ..... 1200 North Walker
LITTLE, JOHN R. ..... Ramsey Tower
LONG, LEROY ..... Medical Arts Bldg.
LONG, LEROY D. ..... Medical Arts Bldg.
LONG, WENDELL ..... Medical Arts Bldg.
LOVE, ROBT. S. ..... Perrine Bldg.
LOWRY, DICK ..... 1200 North Walker
LOWRY, TOM ..... 1200 North Walker
LOY, C. F. ..... Perrine Bldg.
LUTON, JAMES P. ..... Medical Arts Bldg.
McCABE, R. S. ..... Medical Arts Bldg.
MacDONALD, J. C. ..... 300 N.W. Twelfth St.
MARGO, E. ..... 605 N.W. 10th
MARIL, JOS. J. ..... Medical Arts Bldg.
MARTIN, HOWARD C. ..... Ramsey Tower
MARTIN, J. T. ..... 1200 N. Walker
MASTERSON, MAUD M. ..... Medical Arts Bldg.
McBRIDE, EARL D. ..... 605 N.W. 10th
McGEE, J. P. ..... 1200 N. Walker
McHENRY, D. D. ..... Skirvin Tower
McHENRY, L. C. ..... Medical Arts Bldg.
McKINNEY, MILAM F. ..... Medical Arts Bldg.
McNEILL, PHIL M. ..... Medical Arts Bldg.
MECHLING, GEO. S. ..... Osler Bldg.
MELVIN, JAMES H. ..... Perrine Bldg.
MESSENBAUGH, J. F. ..... Colcord Bldg.
MILES, W. H. ..... Municipal Bldg.
MILLER, NESBITT L. ..... Medical Arts Bldg.
MILLS, R. C. ..... Hightower Bldg.
MOORE, C. D. ..... Perrine Bldg.
MOORE, ELLIS ..... Medical Arts Bldg.
MOOR, H. D. ..... 800 N.E. Thirteenth St.
MOORMAN, FLOYD ..... 1200 North Walker
MOORMAN, L. J. ..... 1200 North Walker
MORGAN, C. A. ..... First Natl. Bldg.
MORGAN, VANCE F. ..... State Health Dept.
MORLEDGE, WALKER ..... Osler Bldg.
MORRISON, H. C. ..... 807 North West 23rd St.
MULVEY, BERT M. ..... Medical Arts Bldg.
MURDOCH, RAYMOND L. ..... Medical Arts Bldg.
MUSICK, ELMER R. ..... Medical Arts Bldg.
MUSICK, V. H. ..... Medical Arts Bldg.
MUSSILL, W. M. ..... Medical Arts Bldg.
MYERS, RALPH E. ..... 1200 North Walker
NAGLE, PATRICK S. ..... 1021 No. Lee
NEFF, EVERETT B. ..... 1200 No. Walker
NICHOLSON, B. H. ..... 301 N.W. 12th
NOELL, ROBT. L. ..... Medical Arts Bldg.
O'DONOGHUE, D. H. ..... Medical Arts Bldg.
O'LEARY, CHAS. M. ..... Medical Arts Bldg.
OWEN, CANNON A. ..... 1200 N. Walker
PATTERSON, ROBT. U. ..... 700 N.W. 18th
PAULUS, D. D. ..... 301 West Twelfth St.
PAYTE, J. I. ..... 2429 Aurora Court
PENICK, GRIDER ..... Colcord Bldg.
PHELPS, A. S. ..... Medical Arts Bldg.
PINE, JOHN S. ..... Medical Arts Bldg.
POSTELLE, J. M. ..... Medical Arts Bldg.
POUNDERS, CARROLL M. ..... 1200 North Walker
PRICE, J. S. ..... 1200 North Walker
RECK, J. A. ..... Colcord Bldg.
REED, HORACE, ... Osler Bldg.
REED, JAMES ROBERT ..... Medical Arts Bldg.
REICHMANN, RUTH S. ..... 124 North West 15th St.
RIELY, LEA A. ..... Medical Arts Bldg.
RILEY, J. W. ..... 119 West Fifth St.
ROBINSON, J. H. ..... 301 West Twelfth St.
RODDY, JOHN A. ..... Ramsey Tower
ROGERS, GERALD ..... Osler Bldg.
ROSENBERGER, F. E. ..... Perrine Bldg.
ROUNTREE, C. R. ..... 1200 North Walker
RUCKS, Jr., W. W. ..... 301 West Twelfth St.
RUCKS, W. W. ..... 301 West Twelfth St.
SADLER, LEROY H. ..... Osler Bldg.
SALOMON, A. L. ..... 1200 N. Walker
SANGER, F. A. ..... Key Bldg.
SANGER, F. M. ..... Key Bldg.
SANGER, WINNIE M. ..... Key Bldg.
SARGENT, J. FRANK ..... 320 N.W. 10th
SEIBOLD, GEO. J. ..... Wichita Falls, Texas
SERWER, MILTON J. ..... 1200 North Walker
SEWELL, DAN R. ..... 400 North West Tenth St.
SHELTON, J. W. ..... Medical Arts Bldg.
SHEPPARD, MARY V. S. ..... 1200 North Walker
SHORBE, HOWARD B. ..... Medical Arts Bldg.
SIMON, BETTY HARRIS ..... 1000 N.W. 41st
SMITH, CHAS. A. ..... 719 No. Robinson
SMITH, D. G. ..... First National Bldg.
SMITH, EDW. N. ..... 217 Plaza Court
SMITH, RALPH A. ..... 443½ North West 23rd St.

SNOW, J. B. ..... 1200 North Walker
STANBRO, GREGORY E. ..... Medical Arts Bldg.
STARRY, L. J. ..... 1200 North Walker
STONE, S. N. ..... Edmond
STOUT, M. E. ..... 209 West Thirteenth St.
STRADER, S. ERNEST ..... Hightower Bldg.
STRECKER, W. E. ..... Medical Arts Bldg.
SULLIVAN, ELIJAH S. ..... Medical Arts Bldg.
TABOR, GEO. R. ..... First National Bldg.
TAYLOR, C. B. ..... Medical Arts Bldg.
THOMPSON, W. J. ..... 1200 N. Walker
TOOL, DONOVAN ..... Edmond
TOWNSEND, CARY W. ..... Medical Arts Bldg.
TRENT, ROBT. I. ..... Medical Arts Bldg.
TURNER, HENRY H. ..... 1200 North Walker
UNDERWOOD, E. L. ..... Somerset, Texas
VAHLBERG, E. R. ..... Perrine Bldg.
VON WEDEL, CURT ..... 610 N. W. 9th St.
WAILS, T. G. ..... Medical Arts Bldg.
WAINWRIGHT, TOM L. ..... Medical Arts Bldg.
WARMACK, J. C. ..... 200 N.W. 16th
WATSON, I. N. ..... Edmond
WATSON, O. ALTON ..... 1200 N. Walker
WATSON, R. D. ..... Britton
WEIR, MARSHALL W. ..... Ramsey Tower
WELLS, EVA ..... Medical Arts Bldg.
WELLS, WALTER W. ..... Medical Arts Bldg.
WEST, W. K. ..... 1200 North Walker
WESTFALL, L. M. ..... Medical Arts Bldg.
WHITE, ARTHUR W. ..... Medical Arts Bldg.
WHITE, OSCAR ..... 1200 North Walker
WHITE, PHIL E. ..... Perrine Bldg.
WILDMAN, S. F. ..... Medical Arts Bldg.
WILKINS, HARRY ..... Medical Arts Bldg.
WILLIAMS, L. C. ..... 1200 N. Walker
WILLIAMSON, W. H. ..... 128 N.W. 14th
WILLIE, JAS. A. ..... Medical Arts Bldg.
WILSON, KENNETH J. ..... Medical Arts Bldg.
WOLFF, J. P. ..... 1200 N. Walker
WOODWARD, NEIL W. ..... 1200 North Walker
WRIGHT, HARPER ..... 240 West Commerce
YOUNG, A. M. ..... Medical Arts Bldg.
YOUNG, 3rd, A. M. ..... Medical Arts Bldg.

OKMULGEE

ALEXANDER, LIN ..... Okmulgee
ALEXANDER, ROBT. L. ..... Okmulgee
BOLLINGER, I. W. ..... Henryetta
BOSWELL, H. D. ..... Henryetta
CARLOSS, T. C. ..... Morris
CARNELL, M. D. ..... Okmulgee
COTTERAL, J. R. ..... Henryetta
EDWARDS, J. G. ..... Okmulgee
GLISMANN, M. B. ..... Okmulgee
HOLMES, A. R. ..... Henryetta
HUBBARD, RALPH ..... 1501 E. 11th, Okla. City
HUDSON, W. S. ..... Okmulgee
KILPATRICK, G. A. ..... Henryetta
LESLIE, S. B. ..... Okmulgee
MABEN, CHAS. S. ..... Okmulgee
MATHENEY, J. C. ..... Okmulgee
McKINNEY, G. Y. ..... Henryetta
MING, C. M. ..... Okmulgee
MITCHENER, W. C. ..... Okmulgee
NELSON, J. P. ..... Beggs
RAINS, H. L. ..... Okmulgee
RODDA, E. D. ..... Okmulgee
SIMPSON, N. N. ..... Henryetta
SMITH, C. E. ..... Henryetta
TRACEWELL, GEO. L. ..... Okmulgee
VERNON, W. C. ..... Okmulgee
WALLACE, V M. ..... Morris
WATSON, F. S. ..... Okmulgee

OSAGE

AARON, W. H. ..... Pawhuska
BAYLOR, R. A. ..... Fairfax
DALY, J. F. ..... Pawhuska
DOZIER, B. E. ..... Shidler
ETHERTON, MONTE C. ..... C.C.C. Camp, Pawhuska
GOVAN, T. P. ..... Pawhuska
GUILD, C. H. ..... Shidler
HEMPHILL, G. K. ..... Pawhuska
HEMPHILL, P. H. ..... Pawnuska
KARASEK, M. ..... Shidler
KIMBALL, M. C. ..... Webb City
LIPE, E. N. ..... Fairfax
LOGAN, C. K. ..... Hominy
RAGAN, T. A. ..... Fairfax
RUST, M. E. ..... Pawhuska
SMITH, R. O. ..... Hominy
WALKER, G. I. ..... Hominy
WALKER, ROSCOE ..... Pawnuska
WEIRICH, C. R. ..... Pawhuska
WILLIAMS, CLAUDE W. ..... Pawhuska
WORTEN, D. ..... Pawhuska

OTTAWA

AISENSTADT, E. ALBERT ..... Picher
BARRY, J. R. ..... Picher
CANNON, R. F. ..... Miami
CHESNUT, W. G. ..... Miami
CONNELL, M. A. ..... Picher
CUNNINGHAM, P. J. ..... Miami
DeARMAN, M. M. ..... Miami
DeTAR, G. A. ..... Miami
DOLAN, W. M. ..... Picher
HAMPTON, J. B. ..... Commerce
HETHERINGTON, L. P. ..... Miami
HUGHES, A. R. ..... Afton
KERR, W. C. ..... Picher
LANNING, J. M. ..... Picher
McNAUGHTON, G. P. ..... Miami
MILLER, H. K. ..... Fairland
PURSLEY, TURNER ..... Picher
RALSTON, B. W. ..... Commerce
RITCHEY, H. C.
RUSSELL, RICHARD ..... Picher
SAYLES, W. JACKSON ..... Miami
SHELTON, B. W. ..... Miami
SIEVERS, CHAS. M.
SMITH, W. B. ..... Fairland
WORMINGTON, F. L. ..... Miami

PAWNEE

BERNSTEIN, MAXWELL ..... Pawnee
BROWNING, R. L. ..... Pawnee
HADDOX, C. H. ..... Pawnee
JONES, R. E. ..... Pawnee
LeHEW, ELTON W. ..... Pawnee
LeHEW, J. L. ..... Pawnee
ROBINSON, E. T. ..... Cleveland
SADDORIS, M. L. ..... Cleveland
SPAULDING, H. B. ..... Ralston

PAYNE

BASSETT, C. M. ..... Cushing
CLEVERDON, L. A. ..... Stillwater
DAVIS, BENJ. ..... Cushing
DAVIDSON, W. N. ..... Cushing
FRIEDEMANN, PAUL W. ..... Stillwater
FRY, POWELL E. ..... Stillwater
HACKLER, JOHN F. ..... Stillwater
HARRIS, E. M. ..... Cushing
HERRINGTON, D. J ..... Cushing
HOLBROOK, R. W. ..... Perkins
LEATHEROCK, R. E. ..... Cushing
MANNING, H. C. ..... Cushing
MARTIN, JOHN F. ..... Stillwater
MARTIN, J. W. ..... Cushing
MITCHELL, L. A. ..... Stillwater
PERRY, D. L. ..... Cushing
PUCKETT, HOWARD L. ..... Stillwater
RICHARDSON, P. M. ..... Cushing
ROBERTS, R. E. ..... Stillwater

SMITH, A. B. ..... Stillwater
SMITH, HASKELL ..... Stillwater
STRAHN, EVA ..... Stillwater
THOMPSON, W. C. ..... Stillwater
WAGGONER, ROY E. ..... Stillwater
WALTRIP, J. R. ..... Yale
WILHITE, L. R. ..... Perkins
WRIGHT, J. M. ..... Stillwater

PITTSBURG

BARTHELD, F. T. ..... McAlester
BAUM, F. J. ..... McAlester
COLLINS, GLENN J. ..... McAlester
COOPER, FRED B. ..... McAlester
DORROUGH, JOE ..... Haileyville
GEORGE, L. J. ..... Stuart
GREENBERGER, E. D. ..... McAlester
HARRIS, J. M. ..... Wilburton
KAIESER, WM. H. ..... McAlester
KILPATRICK, G. A. ..... McAlester
KLOTZ, W. F. ..... McAlester
KUYRKENDALL, L. C. ..... McAlester
LIVELY, CLAUDE E. ..... McAlester
McCARLEY, T. H. ..... McAlester
MILLER, F. A. ..... Hartshorne
MUNN, J. A. ..... McAlester
NORRIS, T. T. ..... Krebs
PARK, J. F. ..... McAlester
PEMBERTON, R. K. ..... McAlester
RAMSAY, W. G. ..... Quinton
RICE, O. W. ..... McAlester
RUSSELL, ALLEN R. ..... McAlester
SAMES, W. W. ..... Hartshorne
SHULLER, E. H. ..... McAlester
WAIT, WILL C. ..... McAlester
WELCH, A. J. ..... McAlester
WILLIAMS, C. O. ..... McAlester
WILLOUR, L. S. ..... McAlester
WILSON, HERBERT A. ..... McAlester
WILSON, McCLELLAND ..... McAlester

PONTOTOC

BRECO, J. G. ..... Ada
BRYDIA, CATHERINE ..... Ada
BURROWS, L. I. ..... Ada
CANADA, E. A. ..... Ada
CRAIG, JOHN R. ..... Ada
COWLING, ROBT. E. ..... Ada
CUMMINGS, ISHAM L. ..... Ada
DEAN, W. F. ..... Ada
EVANS, R. ERLE ..... Ada
GULLATT, E. M. ..... Ada
LEWIS, E. F. ..... Ada
LEWIS, MILES L. ..... Ada
McDONALD, GLEN W. ..... Ada
McKEEL, SAM A. ..... Ada
MILLER, OSCAR H. ..... Ada
MOREY, JOHN B. ..... Ada
MUNTZ, E. R. ..... Ada
NEEDHAM, C. F. ..... Ada
PETERSON, WM. G. ..... Ada
ROSS, SAMUEL P. ..... Ada
RUTLEDGE, JAS. A. ..... Ada
SUGG, ALFRED R. ..... Ada
WEBSTER, M. M. ..... Ada
WELBORN, ORANGE E. ..... Ada

POTTAWATOMIE

ANDERSON, R. M. ..... Shawnee
APPLEWHITE, G. H. ..... Shawnee
BAKER, M. A. ..... Shawnee
BALL, W. A. ..... Wanette
BAXTER, GEO. S. ..... Shawnee
BLOUNT, W. T. ..... Durant
BROWN, R. A. ..... Prague
BYRUM, J. M. ..... Shawnee
CAMPBELL, H. G. ..... St. Louis, Okla.
CARSON, F. L. ..... Shawnee
CARSON, JOHN ..... Shawnee
CORDELL, U. S. ..... McComb
CULLUM, J. E. ..... Earlsboro
FORTSON, J. L. ..... Tecumseh
GALLAHER, F. C. ..... Shawnee
GALLAHER, PAUL ..... Shawnee
GALLAHER, W. M. ..... Shawnee
GASTON, JOHN I. ..... Shawnee
GILLICK, D. W. ..... Shawnee
HUGHES, H. E. ..... Shawnee
HUGHES, J. E. ..... Shawnee
IRBY, J. P. ..... Maud
KAYLOR, R. C. ..... McCloud
KEEN, FRANK M. ..... Shawnee
MATTHEWS, W. F. ..... Tecumseh
McADAMS-WILLIAMS, ALPHA ..... Shawnee
McFARLING, A. C. ..... Shawnee
McFARLING, JOHN ..... Shawnee
NEWLIN, FRANCES P. ..... Shawnee
PARAMORE, C. F. ..... Shawnee
RICE, E. E. ..... Shawnee
ROWLAND, T. D. ..... Shawnee
STEVENS, WALTER S., 315 P. O. Bldg., Okla. City
WALKER, J. A. ..... Shawnee
WILLIAMS, A. J. ..... McLoud
STOUGH, A. R. ..... Tecumseh
YOUNG, C. C. ..... Shawnee

PUSHMATAHA

CONNALLY, D. W. ..... Antlers
HUCKABAY, B. M. ..... Antlers
KIRKPATRICK, J. ..... Tuskahoma
LAWSON, JOHN S. ..... Antlers
PATTERSON, E. S. ..... Antlers

ROGERS

ANDERSON, F. A. ..... Claremore
ANDERSON, P. S. ..... Claremore
ANDERSON, W. D. ..... Claremore
BASSMAN, CAROLINE ..... Claremore
BIGLER, EARL E. ..... Claremore
CALDWELL, C. L. ..... Chelsea
COLLINS, B. F. ..... Claremore
HOWARD, W. A. ..... Chelsea
JENNINGS, K. D. ..... Chelsea
MELOY, R. C. ..... Claremore
NELSON, D. C. ..... Claremore

SEMINOLE

CHAMBERS, CLAUDE S. ..... Seminole
DEATON, A. N. ..... Wewoka
GIESEN, A. F. ..... Konawa
HUDDLESTON, W. T. ..... Konawa
LYTLE, WM. R. ..... Seminole
MOSHER, D. D. ..... Seminole
PACE, L. R. ..... Seminole
REEDER, H. M. ..... Konowa
STEVENS, C. ..... Seminole
SHANHOLTZ, MACK I. ..... Wewoka
VAN SANDT, GUY B. ..... Wewoka
VAN SANDT, MAX M. ..... Wewoka

SEQUOYAH

MORROW, J. A. ..... Sallisaw

STEPHENS

COKER, JOHN K. ..... Duncan
GARRETT, S. S. ..... Duncan
HARGROVE, FRED T. ..... Duncan
IVY, W. S. ..... Duncan
KING, E. G. ..... Duncan
LINDLEY, E. C. ..... Duncan
McCLAIN, W. Z. ..... Marlow
McMAHAN, A. M. ..... Duncan
PATTERSON, J. L. ..... Duncan

RICHARDSON, R. W. ..... Duncan
TALLEY, C. N. ..... Marlow
THOMASSON, E. B. ..... Duncan
WALKER, W. K. ..... Marlow
WATERS, CLAUDE B. ..... Duncan
WEEDN, Jr., A. J. ..... Duncan
WILLIAMSON, S. H. ..... Duncan

TEXAS

BLUE, JOHNNY A. ..... Lawton
HAYES, R. B. ..... Guymon
LEE, DANIEL S. ..... Guymon
SMITH, MORRIS ..... Guymon
THURSTON, H. E. ..... Texhoma

TILLMAN

ALLEN, C. C. ..... Frederick
ARRINGTON, J. E. ..... Frederick
BACON, O. G. ..... Frederick
BOX, O. H. ..... Grandfield
CHILDERS, J. E. ..... Tipton
COLLIER, E. K. ..... Tipton
DAVIS, W. W. ..... Davidson
FISHER, R. L. ..... Frederick
FRY, F. P. ..... Frederick
FUQUE, W. A. ..... Grandfield
OSBORN, JR., JAMES D. ..... Frederick
SPURGEON, T. F. ..... Frederick

TULSA

ALLEN, V. K. ..... 1001 Medical Arts Bldg.
ALLISON, T. P. ..... Sand Springs
ARMSTRONG, O. C. ..... 915 Medical Arts Bldg.
AMENT, C. M. ..... 305 Ritz Bldg.
ATCHLEY, R. Q. ..... 507 Medical Arts Bldg.
ATKINS, P. N. ..... Medical Arts Bldg.
BARHAM, J. H. ..... 314 New Daniel Bldg.
BEESLEY, W. W. ..... 501 Medical Arts Bldg.
BEYER, J. W. ..... 621 McBirney Bldg.
BLACK, HAROLD J. ..... 209 Medical Arts Bldg.
BOLTON, J. F. ..... 211 Medical Arts Bldg.
BRADFIELD, S. J. ..... 607 Medical Arts Bldg.
BRADLEY, C. E. ..... 202 Medical Arts Bldg.
BRANLEY, B. L. ..... 315 Med. Arts Bldg.
BRASWELL, JAS. C. ..... 1109 Medical Arts Bldg.
BROGDEN, J. C. ..... 414-15 Medical Arts Bldg.
BROOKSHIRE, J. E. ..... 313 Ritz Bldg.
BROWNE, HENRY S. ..... 615 Medical Arts Bldg.
BRYAN, Jr., W. J. ..... 801 Medical Arts Bldg.
CALHOUN, C. E. ..... Sand Springs
CALHOUN, WALTER H. ..... 405 Daniels Bldg.
CAMERON, PAUL B. ..... 604 S. Cincinnati
CARNEY, A. B. ..... 402 Atlas Life Bldg.
CHALMERS, J. S. ..... Sand Sorings
CHILDS, D. B. ..... 1226 South Boston
CHILDS, HENRY C. ..... 1226 South Boston
CHILDS, J. W. ..... 1226 South Boston
CLINTON, FRED S. ..... 823 Wright Bldg.
CLULOW, GEO. H. ..... 410 McBirney Bldg.
COHENOUR, E. L. ..... 1102 Medical Arts Bldg.
COOK, W. ALBERT ..... 1006 Medical Rrts Bldg.
COULTER, T. B. ..... 1011 Medical Arts Bldg.
CRAWFORD, WM. S. ..... 1228 Exchange Bank Bldg.
CRONK, FRED Y. ..... 801-05 Medical Arts Bldg.
DAILY, R. E. ..... Bixby
DAVIS, B. J. ..... Sand Springs
DAVIS, G. M. ..... Bixby
DAVIS, T. H. ..... 404 Medical Arts Bldg.
DEAN, W. A. ..... 610 Medical Arts Bldg.
DENNY, E. R. ..... 1105 Medical Arts Bldg.
DIEFFENBACH, N. J. ..... 708 Medical Arts Bldg.
DUNLAP, ROY W. ..... 808 Medical Arts Bldg.
EADS, CHAS H. ..... 607 Medical Arts Bldg.
EASON, K. K. ..... 401 Atlas Life Bldg.
EDWARDS, D. L. ..... 203 Philcade Bldg.
EVANS, HUGH J. ..... 303 Medical Arts Bldg.
EWELL, WM. C. ..... 1307 S. Main
FARRIS, H. LEE ..... 303 Medical Arts Bldg.
FLACK, F. L. ..... Natl. Bank of Tulsa Bldg.
FLANAGAN, O. A. ..... 214 Braniff Bldg.
FORD, H. W. ..... 417 Oklahoma Natl. Bank Bldg.
FOREY, W. W. ..... Bixby
FRANKLIN, S. E. ..... Broken Arrow
FULCHER, JOSEPH ..... 417 Medical Arts Bldg.
GARRETT, D. L. ..... 701 Medical Arts Bldg.
GLASS, FRED A. ..... 404 Medical Arts Bldg.
GODDARD, R. K. ..... Skiatook
GOODWIN, SAMUEL ..... 603 Medical Arts Bldg.
GORRELL, J. FRANKLIN, 610 Medical Arts Bldg.
GRAHAM, HUGH C. ..... 1307 So. Main St.
GREEN, HARRY ..... 1116 Medical Arts Bldg.
GROSSHART, PAUL ..... 302 Medical Arts Bldg.
HARALSON, CHAS. H. ..... 816 Medical Arts Bldg.
HARRIS, BUNN, ... Box 356, Jenks
HART, M. M. ..... 1232 South Boulder
HART, M. O. ..... 1232 South Boulder
HAYS, LUVERN ..... Med. Arts Bldg.
HENDERSON, F. W. ..... 304 Medical Arts Bldg.
HENLEY, MARVIN D. ..... Medical Arts Bldg.
HENRY, G. H. ..... 801 Medical Arts Bldg.
HOKE, C. C. ..... 207 Philtower Bldg.
HOOPER, J. S. ..... Woodward
HOOVER, W. D. ..... 201 Philcade Bldg.
HOUSER, M. A. ..... 628 McBurney Bldg.
HOTZ, CARL J. ..... 604 South Cincinnati
HUBER, WALTER A. ..... 1113-14 Medical Arts Bldg.
HUDSON, MARGARET G., 411 Medical Arts Bldg.
HUDSON, DAVID V. ..... 215 Medical Arts Bldg.
HUMPHREY, B. H. ..... Sperry
HUTCHISON, A. ..... Bixby
HYATT, EMRY G. ..... 604 South Cincinnati
JOHNSON, CHAS. D. ..... 1117 Medical Arts Bldg.
JOHNSON, E. O. ..... 206 Medical Arts Bldg.
JOHNSON, R. R. ..... Sand Springs
JONES, W. M. ..... 204 Medical Arts Bldg.
KEMMERLY, H. P. ..... 902 Medical Arts Bldg.
KRAMER, A. C. ..... 415 Medical Arts Bldg.
LARRABEE, W. S. ..... 411 Medical Arts Bldg.
LAYTON, O. E. ..... Collinsville
LeMASTER, D. W. ..... 902 Medical Arts Bldg.
LHEVINE, MORRIS B. ..... 1007 Medical Arts Bldg.
LONEY, W. R. R. ..... 301 Medical Arts Bldg.
LOWE, J. O. ..... 402 Atlas Bldg.
MacDONALD, D. M. ..... 1739 South Utica
MacKENZIE, IAN ..... 511 Medical Arts Bldg.
MARKLAND, JAMES D. ..... Medical Arts Bldg.
MARGOLIN, BERTHA ..... 214 Medical Arts Bldg.
MAYGINNIS, P. H. ..... 505 Palace Bldg.
McCOMB, L. A. ..... 801 Medical Arts Bldg.
McDONALD, J. E. ..... 310 Medical Arts Bldg.
McGILL, RALPH A. ..... 1010 Medical Arts Bldg.
McKELLAR, MALCOLM ..... 604 South Cincinnati
McQUAKER, MOLLY ..... 1648 East 13th St.
MILLER, GEORGE H. ..... 206 Atlas Bldg.
MISHLER, D. L. ..... 604 S. Cincinnati
MOHRMAN, S. S. ..... 604 Daniels Bldg.
MUNDING, L. A. ..... 516 Medical Arts Bldg.
MURDOCK, H. D. ..... 1011 Medical Arts Bldg.
MURRAY, P. G. ..... 506 Medical Arts Bldg.
MURRAY, SILAS ..... 501 Medical Arts Bldg.
MYERS, F. C. ..... 502 Daniels Bldg.
NEAL, JAMES H. ..... 1944 North Denver Place
NELSON, I. A. ..... 1107 Medical Arts Bldg.
NELSON, F. J. ..... 603 Medical Arts Bldg.
NELSON, F. L. ..... Atlas Life Bldg.
NELSON, M. O. ..... 307 Medical Arts Bldg.
NESBITT, E. P. ..... 917 Medical Arts Bldg.
NESBITT, P. P. ..... 917 Medical Arts Bldg.
NORMAN, G. R. ..... 17½ North Lewis
NORTHRUP, L. C. ..... 410 McBirney Bldg.
OSBORN, GEO. R. ..... 1105 Medical Arts Bldg.
PAVY, C. A. ..... 801 Medical Arts Bldg.
PERRY, FRED ..... 804 Atlas Life Bldg.
PERRY, HUGH ..... 804 Atlas Life Bldg.
PERRY, JOHN C. ..... 618 McBirney Bldg.
PIATT, A. D. ..... Medical Arts Bldg.

PIGFORD, A. W. ..... 1001 Medical Arts Bldg.
PIGFORD, CHARLES ..... Medical Arts Bldg.
PIGFORD, R. C. ..... 1001 Medical Arts Bldg.
PITTMAN, COLE D. ..... 1009 Medical Arts Bldg.
PORTER, H. H. ..... 510 Medical Arts Bldg.
PRICE, HARRY ..... 407 Medical Arts Bldg.
RAMEY, CLYDE ..... 612 Palace Bldg.
RAY, R. G. ..... 401 Atlas Bldg.
REECE, K. C. ..... 1101 Medical Arts Bldg.
REYNOLDS, J. L. ..... 305 Palace Bldg.
RHODES, R. E. LEE ..... 509 Medical Arts Bldg.
RICHEY, S. M. ..... 3830 West 44th St.
RILLER, L. E. ..... Mercy Hospital
ROGERS, J. W. ..... 407 Medical Arts Bldg.
ROTH, A. W. ..... 607 Medical Arts Bldg.
RUPRECHT, H. A. ..... 604 South Cincinnati
RUPRECHT, MARCELIA ..... 604 South Cincinnati
RUSHING, F. E. ..... 505 Medical Arts Bldg.
RUSSELL, G. R. ..... 604 South Cincinnati
SCHRECK, PHILIP M. ..... 603 Medical Arts Bldg.
SEARLE, M. J. ..... 202 Medical Arts Bldg.
SHEPARD, R. M. ..... 306 Medical Arts Bldg.
SHEPARD, S. C. ..... 706 Medical Arts Bldg.
SHERWOOD, R. G. ..... 412 Wright Bldg.
SHIPP, J. D. ..... Sisler Clinic
SHOWMAN, W. A. ..... 409 Medical Arts Bldg.
SIMPSON, CARL F. ..... 502 Medical Arts Bldg.
SINCLAIR, F. D. ..... Springer Clinic
SIPPEL, M. E. ..... 1542 East 15th St.
SISLER, WADE ..... 807 South Elgin
SMITH, D. O. ..... 604 South Cincinnati St.
SMITH, N. R. ..... 703 Medical Arts Bldg.
SMITH, ROY L. ..... 809 Medical Arts Bldg.
SMITH, R. N. ..... 1017 Medical Arts Bldg.
SMITH, W. O. ..... 203 Philcade Bldg.
SPANN, L. A. ..... 305 Roberts Bldg.
SPRINGER, M. P. ..... 604 South Cincinnati
STALLINGS, T. W. ..... 724 South Elgin
STEVENSON, JAS. ..... 615 Medical Arts Bldg.
STEWART, H. B. ..... 2500 East 27th Place
STUART, FRANK A. ..... 311 Medical Arts Bldg.
STUART, L. H. ..... 1107 Medical Arts Bldg.
SUMMERS, C. S. ..... 611 Daniels Bldg.
SWANSON, K. F. ..... Springer Clinic
TRAINOR, W. J. ..... 1011 Medical Arts Bldg.
TURNBOW, W. R. ..... 908 Medical Arts Bldg.
UNDERWOOD, D. J. ..... 414-15 Medical Arts Bldg.
UNDERWOOD, F. L. ..... 1001 Medical Arts Bldg.
UNGERMAN, ARNOLD H. ..... 902 Med. Arts Bldg.
VENABLE, S. C. ..... 1135 South Quaker
WALKER, W. A. ..... 322 Kennedy Bldg.
WALLACE, J. E. ..... 914 Medical Arts Bldg.
WALL, G. A. ..... 902 Medical Arts Bldg.
WARD, B. W. ..... 823 Wright Bldg.
WEST, T. H. ..... 612 Medical Arts Bldg.
WHITE, ERIC M. ..... Medical Arts Bldg.
WHITE, N. STUART ..... 416 Medical Arts Bldg.
WHITE, P. C. ..... 312 Medical Arts Bldg.
WILEY, A. RAY ..... 812 Medical Arts Bldg.
WITCHER, R. B. ..... 910 Medical Arts Bldg.
WOODSON, FRED E. ..... 908 Medical Arts Bldg.
ZINK, ROY ..... 807 Daniels Bldg.

WAGONER

BATES, S. R. ..... Wagoner
CRANE, FRANCIS S. ..... Wagoner
PLUNKETT, J. H. ..... Wagoner
RIDDLE, H. K ..... Coweta

WASHINGTON

ATHEY, J. V. ..... Bartlesville
BEECHWOOD, E. E. ..... Bartlesville
CHAMBERLIN, E. M. ..... Bartlesville
CRAWFORD, H. G. ..... Bartlesville
CRAWFORD, J. E. ..... Bartlesville
DORSHEIMER, G. V. ..... Dewey
ETTER, F. S. ..... Bartlesville
GENTRY, RAYMOND C. ..... Bartlesville
GREEN, O. I. ..... Bartlesville
HUDSON, L. D. ..... Dewey
KINGMAN, W. H. ..... Bartlesville
LeBLANC, WM. ..... Ochelata
PARKS, S. M. ..... Bartlesville
REWERTS, F. C. ..... Bartlesville
SHIPMAN, W. H. ..... Bartlesville
SMITH, J. G. ..... Bartlesville
SOMERVILLE, O. S. ..... Bartlesville
STAVER, B. F. ..... Bartlesville
TORREY, J. P. ..... Bartlesville
VANSANT, J. P. ..... Dewey
WEBER, H C. ..... Bartlesville
WEBER, S. G. ..... Bartlesville
WELLS, C. J. ..... Bartlesville
WORD, L. B. ..... Bartlesville

WASHITA

BENNETT, D. W. ..... Sentinel
BUNGARDT, A. H. ..... Cordell
DARNELL, E. E. ..... Colony
HARMS, J. H. ..... Cordell
LIVINGSTON, L. G. ..... Cordell
McMURRY, J. F. ..... Sentinel
NEAL, A. S. ..... Cordell
TRACY, C. M. ..... Sentinel
WEAVER, E. S. ..... Cordell
WEBER, A. ..... Bessie

WOODS

BENJEGERDES, THEODORE D. ..... Beaver
CLAPPER, E. P. ..... Waynoka
DOUGAN, A. L. ..... Amorita
ENSOR, D. B. ..... Hopeton
HALL, RAY L. ..... Waynoka
McGREW, EDWIN A. ..... Beaver
ROGERS, CHAS. L. ..... Carmen
ROYER, CHAS. A. ..... Alva
SIMON, JOHN F. ..... Alva
SIMON, WM. E. ..... Alva
STEPHENSON, ISHMEL F. ..... Alva
STEPHENSON, WALTER LOGAN ..... Aline
TEMPLIN, OSCAR E. ..... Alva
TRAVERSE, CLIFFORD A. ..... Alva

WOODWARD

BEAM, J. P. ..... Arnett
BELL, J. T. ..... Woodward
CAMP, E. F. ..... Buffalo
CHUMLEY, C. P. ..... Supply
DARWIN, D. W. ..... Woodward
DAY, J. L. ..... Supply
DUER, JOE L. ..... Taloga
DUNCAN, J. C. ..... Forgan
ENGLAND, MYRON ..... Woodward
FORNEY, C. J. ..... Woodward
HILL, T. A. ..... Seiling
JOHNSON, H. L. ..... Supply
LEACHMAN, T. C. ..... Woodward
NEWMAN, FLOYD ..... Shattuck
NEWMAN, MESHECH HASKELL ..... Shattuck
NEWMAN, O. C. ..... Shattuck
NEWMAN, ROY ..... Shattuck
RUTHERFORD, V. M. ..... Woodward
SILVERTHORNE, C. R. ..... Woodward
TEDROWE, C. W. ..... Woodward
TRIPLETT, T. B. ..... Mooreland
VINCENT, DUKE W. Vici
WALKER, HARDIN ..... Rosston
WILLIAMS, C. E. ..... Woodward